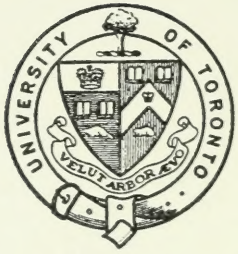

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1989

OPHTHALMOLOGICAL TRANSACTIONS

VOL. XLII

TRANSACTIONS

OF THE

OPHTHALMOLOGICAL SOCIETY

OF THE

UNITED KINGDOM

WITH WHICH ARE AFFILIATED THE

OXFORD OPHTHALMOLOGICAL CONGRESS
MIDLAND OPHTHALMOLOGICAL SOCIETY
NORTH OF ENGLAND OPHTHALMOLOGICAL SOCIETY
IRISH OPHTHALMOLOGICAL SOCIETY
SCOTTISH OPHTHALMIC CLUB
OPHTHALMOLOGICAL SOCIETY OF EGYPT

VOL. XLII

SESSION 1922

WITH

LIST OF OFFICERS, MEMBERS, ETC.

LONDON
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Centralblatt für praktische Augenheilkunde.
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Proceedings, Ophthalmic Section, Royal Society of Medicine.
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Société Belge d'Ophtalmologie.
Transactions of the American Ophthalmological Society.

NOTICE.

The present volume comprises the proceedings of the Ophthalmological Society of the United Kingdom during its Forty-second Session, May, 1922, together with the reports of the proceedings of the Affiliated Societies, 1921–1922.

The Society does not hold itself responsible for the statements, reasonings, or opinions expressed in the communications which the Council has deemed suitable for publication.

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1895 SIR W. R. GOWERS, M.D., F.R.S.
1896 PROF. SNELLEN (Utrecht).

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1902 PROF. E. FUCHS (Vienna).
1904 SIR F. W. MOTT, K.B.E., M.D., F.R.S.
1907 PROF. SATTLER (Leipzig).
1909 EDWARD NETTLESHIP, F.R.S.
1911 E. LANDOLT, M.D. (Paris).
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1921 E. TREACHER COLLINS.

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1907 SIR JOHN HERBERT PARSONS, C.B.E., F.R.S.
1909 EDWARD NETTLESHIP, F.R.S.

ELECTED

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1915 EDWARD TREACHER COLLINS.
1918 GORDON M. HOLMES, C.M.G., C.B.E., M.D.
1921 H. MOSS TRAQUAIR, M.D.

*** *Members are requested to communicate with the Council's Secretary when corrections are necessary.*

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EXPLANATION OF ABBREVIATIONS.

O.M.—Original Member.
Pres.—President.
V.-P.—Vice-President.
T.—Treasurer.
L.—Librarian.
S.—Secretary.
C.—Member of Council.
*.—Denotes Resident Life Members.
†.—Denotes Non-Resident Life Members.

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ELECTED

1905 ADAMS, P. E. H., M.B., The Cardinal's Hat, 6, Holywell, Oxford.
1912 ALEXANDER, G. F., M.B., 35, Prince of Wales Terrace, Scarborough.
1907 ALLPORT, WILFRED, M.B., 95, Cornwall Street, Birmingham.
1899 †AMENABAR, DANIEL, Casiila 699, Santiago, Chili.
1910 ANGELUCCI, Prof., 2, Piazza N. Amore, Naples.
1913 ANKLESARIA, M. DHANJISHAW, Ahmedabad, India.
1891 †ARMSTRONG, HUGH, Hobart, Tasmania.
1895 ATTLEE, JOHN, M.D., 65, Grosvenor Street, W. 1.

ELECTED

1922 Bainbridge, William, M.B., Ch.B., 7, Albion Street, Hull.

1903 Ballantyne, A. J., M.D., 11, Sandyford Place, Glasgow. (C. 1914–17.)

1922 Baranov, M., M.D., B.S., 30, York Street, Johannesburg, S. Africa.

1907 Bardsley, Percy C., M.B., 67, Wimpole St., W. 1.

O.M. *Barlow, Sir Thomas, Bart., K.C.V.O., M.D., 10, Wimpole Street, W. 1. (C. 1880-81. V.-P. 1894-7.)

1920 Barradah, Hassam, P.M.O., Ophthalmic Hospital, Sohag, Egypt.

1889 †Barrett, Sir James W., K.B.E., C.B., C.M.G., M.D., 34, Collins Street East, Melbourne, Australia.

1917 Barrie, T. Stewart, M.B., 13, Somerset Place, Sauchiehall Street, Glasgow.

1920 Barton, Maurice H., 27, Horse Fair Street, Leicester.

1922 Batt, Bernard E. A., M.B., B.Ch., 6, Angel Hill, Bury St. Edmunds.

1892 Batten, Rayner D., M.D., 81, Harley Street, W. 1. (C. 1905-8.)

1888 *Beaumont, W. M., 4, Gay Street, Bath. (V.P. 1912–15. C. 1901-4.)

1912 Beedham, Henry Wm., M.B., 15, Crediton Hill, West Hampstead, N.W. 6.

1891 †Bennett, Alfred H., 16, North Terrace, Adelaide, South Australia.

1897 Bennett, H. Percy, M.B., 12, Victoria Square, Newcastle-on-Tyne.

1905 Bennett, F. D., M.D., 18, Savile Row, W. 1.

O.M. Berry, Sir George A., LL.D., M.B., 31, Drumsheugh Gardens, Edinburgh. (*Pres.* 1909-11. C. 1889-92. V.-P. 1895-8. 1912.)

1899 Bickerton, R. E., D.S.O., M.B., 137, Harley Street, W. 1.

1881 *Bickerton, T. H., 88, Rodney Street, Liverpool. (C. 1895-8. V.-P. 1910-12.)

1905 Bird, J. W., D.S.O., Egham Hill, Egham, Surrey.

ELECTED

1892 Black, John Wilson, M.D., 46, Academy Street, Inverness.

1919 Blair, C. J. S., 51, Sheen Road, Richmond.

1898 Blair, Charles Samuel, M.D., 14, Welbeck Street, W. 1.

1917 Bodkin, Herbert, 4, Mutual Buildings, Maitland Street, Bloemfontein, O.F.S., South Africa.

1895 †Bonar, Thomson, M.D., "Glenview," Colinton, Midlothian.

1907 Bond, C. Shaw, M.B., Liphook, Hants.

1911 Boucher, R. B., M.D., Vancouver, B.C.

1885 Bower, Ernest Dykes, M.D., Elton House, Gloucester.

1897 Bower, John A., M.B., 18, Royal Crescent, Cheltenham.

1905 Brailey, A. R. (C.), 24, Harley Street, W. 1.

1902 *Brailey, Wm. Herbert, M.D., 21, Lansdowne Place, Hove, Sussex.

1921 Bransgrove, William S. V., M.B., c/o Bank of New Zealand, 1, Queen Victoria Street, E.C. 4.

1911 Brearley, E. A., M.B., 141, Macquarie Street, Sydney, N.S.W.

1911 Brennard, H. T. W., University Club, Sydney, N.S.W.

1899 Brewerton, Elmore, 73, Harley Street, W. 1. (C. 1909–10; 1914–17. S. 1910-13.)

1920 Bride, T. Milnes, M.D., 26, St. John Street, Manchester.

1903 Brinton, A. G., Box 5852, Johannesburg, South Africa.

1909 Broachâ, B. B., Karachi Eye Hospital, Karachi, India.

1886 Bronner, Adolph, M.D., 33, Manor Row, Bradford. (C. 1900–3.)

1901 *Brooks, R. Philip, 14, Welbeck Street, W. 1.

1895 Browne, Sir J. Walton, M.D., 10, College Square North, Belfast.

1922 Browning, Sidney Howard, 22, Harley Street, W. 1.

1901 Buchanan, Leslie, 8, Royal Crescent West, Glasgow.

1907 Buckland, Francis, M.D., Moorland Court, Poole Road, Bournemouth.

ELECTED

1907 Burdon-Cooper, J., M.D., 12, The Circus, Bath.

O.M. †Burnham, G. Herbert, M.D., 128, Bloor Street West, Toronto, Canada.

1909 Butler, T. Harrison, M.D., B.Ch. (C.), 26, Adelaide Road, Leamington.

1902 Buzzard, E. Farquhar, M.D., 78, Wimpole Street, W. 1. (C. 1908-9. S. 1909-12.)

1897 †Byers, W. Gordon M., M.D., 346, Mountain Street, Montreal.

1892 †Caiger, Herbert, M.B., 387, Glossop Road, Sheffield.

1922 Caleb, C. C., M.B., M.S., Westfield, 34, Winchester Road, Worthing.

1905 Campbell, C. A., 172, Bloor Street East, Toronto.

1891 Cargill, L. Vernon, 35, Cavendish Square, W. 1. (C. 1905-8.)

1921 Carruthers, J. Ferguson, M.D., Royal Herbert Hospital, Woolwich, S.E. 18.

1885 Clarke, Ernest, M.D., B.S., 3, Chandos Street, Cavendish Square, W. 1. (C. 1893-6. V.-P. 1910-12.)

1911 Clegg, John Gray, M.D., 22, St. John Street, Manchester.

1905 Clements, E. C., 3, Lindum Road, Lincoln.

1907 Cobbledick, Arthur Stanley, M.D., B.S., 21, Bolton Street, Mayfair, W. 1.

1919 Cohen, Louis David, 200, Willis Street, Wellington, New Zealand.

1885 Collins, E. Treacher, 17, Queen Anne Street, W. 1. (*Pres.* 1917-18. V.-P. 1905-8; 1916-17; 1918–21. C. 1889-92; 1901-4. S. 1898-1901.)

1886 Collins, Sir W. J., K.C.V.O., M.S., M.D., 1, Albert Terrace, Gloucester Gate, Regent's Park, N.W. 1.

1920 Conway, James A., M.D., 337, Bath Street, Glasgow, W.

1901 Cooke, Arthur, M.B., Grove Lodge, Cambridge.

1901 Cooper, Ludford, M.D., 16, Star Hill, Rochester, Kent.

1894 †Coote, Patrick, M.D., 73, St. Ann Street, Quebec, Canada.

ELECTED

1921 Coppinger, Lieut.-Col. W. V., D.S.O., M.D., I.M.S., 6, Little Russell Street, Calcutta.

1904 Corbett, W. J., 18, Weymouth Street, W. 1.

1921 Cormack, Capt. H. S., M.C., M.B., I.M.S., c/o Messrs. Grindlay & Co., Parliament Street, London, S.W. 1.

1898 Coulter, Robert J., M.B., Ivor House, Gold Tops, Newport, Mon.

1895 †Courtenay, J. D., M.D., Ottawa, Canada.

1909 Craig, James A., M.B. 11, University Square, Belfast. (C. 1915–17.)

1901 Crawley, F. C., M.D., 5, Fitzwilliam Place, Dublin.

1907 Cree, Robert Ewart, M.D., 40, Jesmond Road, Newcastle-on-Tyne.

1899 Cresswell, F. P. S., M.B., B.S., 24, Windsor Place, Cardiff.

1922 Cresswell, Thomas H., 3, Lindum Road, Lincoln.

1904 Cridland, A. Bernard, Salisbury House, Chapel Ash, Wolverhampton. (C. 1918–21.)

O.M. Critchett, Sir G. Anderson, Bart., K.C.V.O., 21, Harley Street, W. 1. (*Pres.* 1899-1901. C. 1883-6. V.-P. 1894-7, 1902-5.)

1898 Critchley, Harry G., M.D., 83, Park Lane, Croydon.

1906 Crocket, A. Pierce. (Address uncommunicated.)

1881 Cross, F. R., M.B., LL.D., Worcester House, Clifton, Bristol. (*Pres.* 1913–15. V.-P. 1898-1901, 1915-17. C. 1891-4.)

1902 Cruise, Sir R. R., K.C.V.O. (C. 1917-20), 34, Wimpole Street, W. 1.

1912 Cummins, J. D., M.B., 10, Clare Street, Dublin.

1904 Cunningham, H. H. B., M.D., Lt.-Col. R.A.M.C., c/o P. D. S. Cunningham, Esq., 3308, Ewart Street, New Westminster, B.C., Canada.

1905 Cunningham, J. F., O.B.E. (S.), 27, Weymouth Street, W. 1. (C. 1917-20).

1921 Cursetji, Capt. H. R., I.M.S., c/o Thomas Cook & Sons, Bombay.

ELECTED

1916 Danis, Marcel, M.D., Rue de la loi 134, Brussels, Belgium.

1922 Davenport, Robert Cecil, M.B., B.S., St. Bartholomew's Hospital, E.C. 1.

1907 Davidson, Alex. Dyce, M.B., "Greenfield," Archway Road, Highgate, N. 6.

1917 Davidson, Isaac A., M.D., 3, Laurington, Antrim Road, Belfast.

1903 Davies, David, M.B., 8, Lonsdale Gardens, Tunbridge Wells.

1908 Davies, D. L., M.S., M.D., 31, Newport Road, Cardiff.

1914 De Lapersonne, Prof., M.D., 30 Rue de Lisbonne, Paris.

1903 Dean, C. W., Lindow Square, Lancaster.

1903 Dickson, R. H., Nelson Place, Newcastle-under-Lyme, Staffordshire.

1910 Dighton, Chas. A. Adair, M.B., 73, Rodney Street, Liverpool.

1881 Dixon, W. E., "Kirkee," Mill Road, West Worthing.

1905 Dorrell, E. Arthur, Bardsey, 19, Bath Road, Reading.

1920 Doyne, P. G., 8, Harley Street, W. 1.

1889 †Drake-Brockman, Herbert E., Col. I.M.S., c/o Thomas Cook and Son, Ludgate Circus, E.C. 4.

1895 Du Boulay, H. H., 2, Royal Terrace, Weymouth.

1909 Duggan, J. N., Sir J. C. Ophthalmic Hospital, Girguina, Bombay.

1900 Eason, H. L., C.B., C.M.G., M.B., 62, Portland Place, W. 1. (C. 1915–17.)

1919 East, A. Gerard, M.B., Southleigh, Truro.

O.M.*Edmunds, Walter, M.B., 2, Devonshire Place, Portland Place, W. 1. (C. 1885-8.)

1902 †Elliot, Robert Henry, Lt.-Col. I.M.S. (retired), M.D., Sc.D., 54, Welbeck Street, W. 1.

1903 Ellis, W. F., c/o Messrs. Holt, 3, Whitehall Place, S.W. 1.

1921 Eminson, C. F., The Royal Infirmary, Sheffield.

1908 Emmerson, Herbert H., 275, Glossop Road, Sheffield.

1883 *Emrys-Jones, A., M.D., 10, St. John Street, Manchester.

ELECTED

1918 Evans, Frederick Hudson, The Glen, 161, Lewisham Road, S.E. 13.

1900 Evans, J. Jameson, M.D., 41, Newhall Street, Birmingham.

1907 Fenton, Lt.-Col. A., M.B., I.M.S., c/o Messrs. Thos. Cook and Son, Rangoon.

1911 Fenwick, George, O.B.E., Northern Club, Auckland, New Zealand.

1888 Fergus, A. Freeland, M.B., 14, Newton Place, Charing Cross, Glasgow. (C. 1905-8.)

O.M. †Ferguson, H. L., M.D., Dunedin, New Zealand.

1894 †Fischer, E. C., M.D., Cairo, Egypt.

1904 Fisher, Lt.-Col. J., D.S.O., I.M.S., c/o Messrs. Grindlay and Co., 54, Parliament Street, S.W. 1.

1895 Fisher, J. Herbert, M.B., B.S. (V.-P.), 83, Wimpole Street, W. 1. (C. 1904-7; 1911-12; S. 1907-10; V.-P. 1918-20; *Pres.* 1920–22.)

1919 Fison, James, M.D., 9, North Park Road, Harrogate.

1912 Flaxman, Samuel C. R.

1917 Fleming, Norman B. R., M.B., 92, Harley Street, W. 1.

1889 Flemming, Percy, M.D., 70, Harley Street, W.1. (V.-P. 1911–13; C. 1901-4.)

1892 Folker, Herbert Henry, O.B.E., Bedford House, Shelton, Stoke-on-Trent.

1919 Ford, Rosa, M.B., 46, Bernard Street, Russell Square, W.C. 1.

O.M. Frost, W. Adams. (Address uncommunicated.) (L. 1900-6. V.-P. 1906-9.)

1895 Galloway, A. Rudolf, O.B.E., M.B., 250, Union Street, Aberdeen.

1887 †Gardner, John J., M.D., 128, Bishop Street, Montreal, Canada.

1920 Gardner, Mark C., M.D., Box 252, G.P.O., Melbourne, Australia.

1913 Ghaswala, K. S., The Eye Hospital, opposite Marine Lines Station, Queen's Road, Bombay.

1912 Gibb, H. P., M.B., 91, Harley Street, W. 1.

ELECTED

1889 Gibbs, Alfred N. G., 52, Whiteladies Road, Clifton.

1902 †Gibson, J. Lockhart, M.D., Wickham Terrace, Brisbane, Australia.

1921 Gilmour, H. M., 22, Museum Street, Ipswich.

1918 Girgis, A. M., Assiut, Egypt.

1918 Girgis, B., Ophthalmic Hospital, Cairo.

1917 Giri, D. V., c/o Coutts & Co., 440, Strand, W.C. 2.

1899 Goldsmith, G. Harvey, M.B., Harpur Place, Bedford.

1907 Goulden, Charles Bernard, O.B.E., M.D., M.C. (C.), 42, Welbeck Street, W. 1.

1904 Gowans, Thos., M.B., Henley Grange, Great Smeeton, near Northallerton.

1897 Granger, F. M., 18, Nicholas Street, Chester.

1895 †Grant, H. Y., M.D., 414, Delaware Avenue, Buffalo, U.S.A.

1922 Graves, Basil, M.C., 35, Weymouth Street, W. 1.

1895 †Gray, James, M.D., 119, Robert Street, Toronto, Canada.

1887 Green, Edwin Collier, 27, Friar Gate, Derby. (C. 1903-4.)

1903 Greene, Arthur, M.D., 4, Theatre Street, Norwich.

1911 Greeves, Reginald A., M.B., 23, Wimpole Street, W. 1.

1885 *Griffith, A. Hill, M.D., Minbridge House, near Woking, Surrey. (V.-P. 1905-8. C. 1893-6.)

1910 Griffith, Arthur Donald, M.B., 39, Hertford Street, Mayfair, W. 1.

1894 Grimsdale, Harold, M.B. (V.-P.), 3, Harley Place, Marylebone Road, N.W. 1. (C. 1907-10.)

1899 Gruber, Rudolph, M.D., 81, Harley Street, W. 1.

1908 Gunning, C. J. H., 50, Brook Street, Grosvenor Square, W. 1.

1910 Haab, Prof. O., 41, Pelikanstrasse, Zurich, Switzerland.

1889 Hailes, C. D. G., M.D., 2, Richmond Park Road, Clifton, Bristol.

1885 †Haines, Humphrey, Auckland, New Zealand.

ELECTED

1902 †Halliday, J. C., M.B., Macquarie Street, Sydney, N.S.W.

1900 Hallidie, Andrew, M.B., 7, Grosvenor Crescent, St. Leonard's-on-Sea.

1912 Hanafin, Major P. J., D.S.O., R.A.M.C. (Address uncommunicated.)

1901 Hancock, Edw. D., Merrow Grange, Guildford.

1908 Hanna, Henry, M.B., 30, University Square, Belfast.

1901 Hanson, Reginald E., M.B., 20, Kensington Park Gardens, W. 11.

1920 Harford, C. F., M.D., 30, Finsbury Square, E.C. 2.

1900 Harman, N. B., M.B., 108, Harley Street, W. 1.

1902 Harries-Jones, E., M.D., 16, Castilian Street, Northampton.

1910 Harston, George M., M.D., Hong Kong.

1882 *Hartridge, Gustavus, 12, Wimpole Street, W. 1. (C. 1889-92, 1895-8. S. 1892-5. V.-P. 1910-11.)

1905 †Harty, G. W., M.B., Ch.B., Wellington, New Zealand.

1899 Hawkes, C. S.

1901 Hawthorne, C. O., M.D., 63, Harley Street, W. 1.

1908 Hay, Percival J., M.D., 350, Glossop Road, Sheffield.

1892 Haydon, Frank, Apothecaries' Hall, Water Lane, Blackfriars, E.C. 4.

1921 Healy, James Joseph, M.B., Lucania Buildings, Llanelly, South Wales.

1899 Henderson, Edward E., M.B., Redrigg, Ferry Road, Felixstowe.

1903 Henderson, Thomson, M.D., 15, The Ropewalk, Nottingham.

1896 Henry, R. Wallace, M.D., 6, Market Street, Leicester.

1905 Hepburn, Malcolm Langton, M.D., 111, Harley Street, W. 1.

1897 Herbert, Lt.-Col. Herbert, I.M.S. (retired), 19, Brunswick Place, Hove, Sussex. (C. 1911–13.)

1887 *Hern, John, M.D., Semmercote, Darlington.

1910 Heron, David. Burton Court, Chelsea, S.W. 3.

ELECTED

1905 Hewkley, F., 72, Wickham Road, Brockley, S.E. 4.

1900 Heyliger, E. D. Scott, M.B., C.M., 89, Preston New Road, Blackburn.

1895 Hickman, H. R. Belcher, M.B., Castle House, Flux Bourton, near Bristol.

1920 Highet, H. C., M.D., Radley House, Winchester.

1916 Hine, Montague, M.D., 30, Weymouth Street, W. 1.

1888 *Hinnell, J. S., M.D., 62, Garland Street, Bury St. Edmunds.

1905 Hird, R. B., M.D., 52, Newhall Street, Birmingham.

1919 Hodge, G., M.D., 603, Dorchester Street West, Montreal.

1897 Hogg, G. H., M.D., 37, Brisbane Street, Launceston, Tasmania.

1911 Holmes, Gordon M., C.M.G., C.B.E., M.D., 101, Harley Street, W. 1. (S. 1912–15, C. 1915–17.)

1889 Holthouse, Edwin H., M.B., 35, Nottingham Place, W. 1.

1917 Holyoak, W. L., M.D., 45, Welbeck Street, W. 1.

1905 Hosford, J. S., 31, Weymouth Street, W. 1.

1893 †Howe, Lucien, 520, Delaware Avenue, Buffalo, N.Y., U.S.A.

1908 Hudson, A. C., M.D., 50, Queen Anne Street, W. 1.

1889 †Hughes, Samuel H., 173, Macquarie Street, Sydney, New South Wales.

1893 †Hughes, Wilfrid Kent, M.B., 102, Collins Street, Melbourne.

1908 Hunter, George, 31, Bridge Street, Inverness.

1919 Husain, T., Khan-Bahadur Eye Hospital, Allahabad, India.

1921 Ingle, E. W., M.D., Davenport Road, Durban, Natal, S. Africa.

1922 Iles, Arthur Ernest, M.B., B.S., 17, Victoria Square, Clifton, Bristol.

1901 Inman, Wm., M.B., 22, Clarendon Road, Southsea.

1898 James, George Brooksbank, 5, Harley Street, W. 1. (C. 1911–13.)

ELECTED

1908 James, Robert Rutson (C.), 46, Wimpole Street, W. 1. (S. 1918–21.)

1905 Jaques, R., 20, Athenæum Street, Plymouth.

1918 Jellett, James William Henry, M.D., Dublin, 11, Beresford Street, Waterford.

1883 †Jenkins, E. J., M.D., Nepean Towers, Douglass Park, Sydney, N.S.W., Australia.

1908 Jeremy, H. Rowe, 66, Harley Street, W. 1.

1910 Jewell, William H., M.D., 27, Queen Anne Street, W. 1.

1882 Johnson, G. Lindsay, M.D., Britannia Buildings, West Street, Durban, Natal.

1888 †Johnston, Geo. D., Georgia Street, Vancouver, British Columbia.

1898 *Jones, George, M.B., 8, Church Terrace, Lee, S.E. 13.

1897 Jones, Hugh E., 19, Rodney Street, Liverpool.

1894 †Jones, R. H., M.B., B.S., 209, Macquarie Street, Sydney, New South Wales.

1914 Joseph, Hugh Percival, Eye Hospital, Colombo, Ceylon.

1905 Juler, F. A., M.B., B.C. (C.), 14, Portland Place, W. 1. (S. 1919–22.)

1914 Karani, N. N., "Eye Clinic," near Edward Theatre, 18, Kalbadevi Road, Bombay.

1899 Keeling, G. S., M.D., Attleborough, Norfolk.

1900 †Kelsall, H. T., M.D., 1, Devonshire Terrace, Perth, W. Australia.

1898 †Kendall, H. W. Martindale, Wellington, New Zealand.

1888 †Kenny, Augustus Leo, M.B., Ch.B., 13, Collins Street, Melbourne, Victoria, Australia.

1921 Kiep, Walter H., M.B., Kaser-el-Ami Hospital, Cairo, Egypt.

1913 Killen, W. M., M.D., 32, College Gardens, Belfast.

1904 Killick, Chas., M.D., 14, Spring Bank, Manningham Lane, Bradford.

ELECTED

1906 Kilkelly, Col. P. P., I.M.S. (retired). c/o Messrs. H. S. King and Co., Pall Mall, S.W. 1.

1922 Kirkpatrick, Henry, M.B. (Lieut.-Col. I.M.S.), 54, Welbeck Street, W. 1.

1895 Knaggs, Robert Lawford, M.D., 27, Park Square, Leeds.

1910 Knapp, Arnold, M.D., 10, East 54th Street, New York, U.S.A.

1911 Lang, B. T., B.C., 22, Cavendish Square, W. 1.

O.M. Lang, William, 22, Cavendish Square, W. 1. (C. 1886-9. V.-P. 1900-3 (*Acting Pres.* 1903.)

O.M. Lawford, J. B., M.D., LL.D., 27, Weymouth Street, W. 1. (C. 1886-9; 1898-1901. S. 1895-8. V.-P. 1905; 1913-16. T. 1905-11. *Pres.* 1911-13.)

1889. *Laws, Wm. G., M.B., 3, East Circus Street, Nottingham. (C. 1909-11.)

1896 Lawson, Sir Arnold, K.B.E. (T.), 12, Harley Street, W. 1. (V.-P. 1917. C. 1909-1911.)

1895 †Lea, J. Augustus, M.B., Grahamstown, Cape Province, S. Africa.

1885† Le Cronier, Hardwick, St. Heliers, Jersey.

1917 Leask, H. L. G., M.D., 70, Dixon Avenue, Crosshill, Glasgow.

O.M. Lediard, H. A., M.D., 26, Lowther Street, Carlisle. (C. 1900-1.)

1912 Lee, H., M.B., 6, Park Square, Leeds.

1904. †Lee, W. A., Col. I.M.S. (retired), 44, Primrose Hill Road, N.W. 3.

1911 Letchworth, T. W., M.B., 68, Claremont Road, Surbiton.

1903 Levy, A., M.D., 67, Wimpole Street, W. 1.

1907 Lindsay, W. J., M.D., 84, Herne Hill, S.E. 24.

1921 Lister, Major A. E. J., I.M.S., c/o Lloyds Bank, Dursley, Gloucestershire.

1896 Lister, Sir W. T., K.C.M.G., M.D., 24, Devonshire Place, W. 1. (V.-P. 1918-21. T. 1921-22. C. 1907-10.)

1892 Lodge, Samuel, jun., M.D., 28, Manor Road, Bradford.

ELECTED

1911 Loosely, Alfred E. A., M.B., 25, New Cavendish Street, W. 1.

1903 †Lückhoff, James, M.D., Rhodes Buildings, St. George's Street, Capetown.

1899 Lyle, H. Willoughby, M.D., Speldhurst, Elmfield Road, Bromley, Kent.

1900 MacCallan, Arthur Ferguson, C.B.E., M.B., Public Health Department, Cairo, Egypt.

1913 McCready, W., M.B., 2, University Square, Belfast.

1918 McCulloch, R. J. P., M.B., L.R.C.P., 102, College Street, Toronto, Canada.

1920 MacDonald, Alexander, M.B., c/o J. McWhirter, Esq., The Valley, Brisbane, Queensland.

1905 Macdonald, J. G., Imperial Chambers, Dee Street, Invercargill, New Zealand.

1919 MacFetridge, William C., M.D., 27, Upper Fitzwilliam Street, Dublin.

1920 MacGillivray, Allister M., M.B., 23, South Tay Street, Dundee.

1890 *MacGillivray, Angus, M.D., 23, South Tay Street, Dundee. (C. 1902-5.)

1902 Mackay, D. Matheson, M.D., 15, Albion Street, Hull.

1888 *Mackay, George, M.D., 26, Drumsheugh Gardens, Edinburgh. (C. 1900-2. V.-P. 1906-9.)

1889 †Mackenzie, F. Wallace, M.B., 139, Upper Willis Street, Wellington, New Zealand

1895 McKenzie, H. V., M.D., Elmbank, Abbey Road, Torquay.

1889 MacLehose, Norman M., M.B., 18, Harley Street, W. 1. (C. 1902-5.)

1897 †Maclennan, Duncan N., M.D., 126, Bloor Street West, Toronto.

1892 †MacLeod, Charles G., M.D., 157, Macquarie Street, Sydney, N.S.W.

1922 Maher, Herbert Odillo, M.B., Ch.M., 195, Macquarie Street, Sydney, N.S.W.

1922 Mann, Ida Caroline, M.B., 13, Minster Road, Cricklewood, N.W. 2.

ELECTED

1902 McMullen, W. H., M.B., O.B.E., 133, Harley Street, W. 1. (C. 1914-17.)

1911 McNabb, Henry H., M.D., 84, St. John Street, Manchester.

1918 MacNeil, Capt. Frank A., 810, Boyd Buildings, Winnipeg, Canada.

1912 McPherson, Major, I.M.S., c/o Ophthalmic Hospital, Bombay, India.

1911 MacQueen, J. F.,

1899 Maddox, Ernest E., M.D., Glenartney, Poole Hill, Bournemouth. (C. 1904-6.)

1918 Maghy, Charles Alfred, Suite 409, Timken Building, San Diego, California, U.S.A.

1922 Maher, H. Odillo, 195, Macquaire Street, Sydney, N.S. Wales, Australia.

1899 †Manché, Charles, B.A., M.D., 33, Strada Alessandro, Valletta, Malta.

1922 Mann, Ida C., M.B., B.S., 13, Minster Road, Cricklewood, N.W. 2.

1901 †Manning, Leslie S., M.B., Christchurch, New Zealand.

1911 Manson, W. H., M.D., 17, Royal Terrace, Glasgow, W.

1904 Markus, Charles, M.D., 28, Wimpole Street, W. 1.

1883 †Marlow, Frank William, M.D., 200, High Land Street, Syracuse, N.Y., U.S.A.

1916 Marquez, M., Dr., Puerta del Sol, 13, Madrid, Spain.

1907 Marshall, James Cole, M.D., 126, Harley Street, W. 1.

1888 †Martin, Albert, M.D., Wellington, New Zealand.

1919 Mason, Mrs. Mary E. Joll, M.D., 25, Gordon Square, W.C. 1.

1905 Massey, A. Vale, M.D., 24, Tavistock Square, W.C. 1.

1918 Mathieu, Capt. Albert, C.A.M.C. (Address uncommunicated).

1918 Matthews, Richard H., M.D., 4, Upper Fitzwilliam Street, Dublin.

1916 Maxted, George, M.D., 14, Thorpe Mansions, Norwich.

1914 Maxwell, Miss E. M., M.B., 19, Lower Baggot Street, Dublin.

ELECTED

1904 May, H. J., M.B., B.C., 21, The Avenue, Southampton.

1902 Mayou, Stephen, 59, Harley Street, W. 1. (C. 1912–15, 1913–22. S. 1916–19.)

1912 Mead, John Clarke, M.B., "Bryn-y-mor," Yarmouth Road, Lowestoft.

1901 Menzies, J. A., M.B., 9, Castle Hill Avenue, Folkestone.

1920 Miller, H. P., M.D., 100, Stoke Newington Road, N. 16.

1897 †Minnes, Robert Stanley, M.D., 127, Metcalfe Street, Ottawa, Ontario.

1921 Mishra, R. S., M.B., B.S., Rutlam, Central India.

1911 Mitchell, Leonard J. C., M.D., 4, Collins Street, Melbourne.

1921 Moodie, A. R., M.B., 5, Windsor Terrace, Dundee.

1896 Mooney, Herbert C., M.B., 33, Fitzwilliam Place, Dublin.

1908 Moore, R. Foster, O.B.E., M.B., B.C. (S.), 91, Harley Street, W. 1.

1909 Morrell, Reginald A., Headington Orthopaedic Hospital, Headington, Oxford; 6, King Edward Street, Oxford.

1896 Mott, Sir F. W., K.B.E., M.D., F.R.S., 25, Nottingham, Place, W. 1. (V.-P. 1904–7.)

1908 Moxon, Frank, O.B.E., M.B., B.S., 4, Bentinck Street, W. 1.

1921 Mukerjee, S. K., 13, Kyd Street, Calcutta.

1916 Mulholland, P. J. H., 5, University Terrace, Belfast.

1909 Naylor, W. Roebuck, 14, Carr Road, Nelson, Lancs.

1920 Neame, Humphrey, 256, Temple Chambers, Temple Avenue, E.C. 4.

1909 Nell, Andreas, Victoria Eye and Ear Hospital, Colombo, Ceylon.

1881 Nicholson, A., M.B., 30, Brunswick Square, Brighton.

1910 Nordenson, Dr. Erik, 10, Wasagatan, Stockholm, Sweden.

1912 Norman, Alfred Clarence, M.D., X-ray Institute, New General Hospital, Baghdad, Mesopotamia. *Trans.* to The Glen, Stonehouse, Glos.

ELECTED

1889 †O'Kinealy, Frederick, C.I.E., C.V.O., Lt.-Col., I.M.S., c/o Messrs. H. S. King & Co., 9, Pall Mall, S.W. 1.

1911 Oliver, M. W. B., O.B.E., M.B., 128, Harley Street, W. 1.

1914 Oliver George H., 284, Manningham Lane, Bradford.

1899 Ormond, Arthur Wm., C.B.E., 7, Devonshire Place, W. 1. (C. 1910-12).

1892 †Orr, Andrew William, M.D., Brisbane.

1890 †Osborne, A. B., M.D., 46, McNab Street South, Hamilton, Canada.

1890 †Palmer, L. Loran, M.D. (Address uncommunicated.)

1894 *Parker, Herbert, Saragossa House, New Street, Henley-on-Thames.

1899 Parker, Herbert George, 13, Chorley New Road, Bolton.

1900 Parsons, Sir John Herbert, C.B.E., F.R.S., M.B., D.Sc., (V.-P.), 54, Queen Anne Street, W.1. (C. 1909-12.)

1887 †Patel, D. H., Bai Hirabai, B. J. Charitable Dispensary Tardeo, Bombay.

1907 Paterson, James V., M.B., 11, Melville Street, Edinburgh.

1900 †Patkar, Bhagvant Sakharam.

1902 Paton, Leslie, M.B., 29, Harley Street, W. 1. (C. 1912–15.)

1918 Pearse, Patrick, L.R.C.P., L.R.C.S., 17, The Crescent, Limerick.

1888 Percival, Archibald Stanley, M.B., B.Ch., Westward, Tankerville Terrace, Newcastle-on-Tyne. (C. 1906-9.)

1891 Perry, Sir A., M.D., Surgeon-Major, Principal Civil Medical Officer, Colombo, Ceylon.

1889 †Perry, Francis F. (Address uncommunicated.)

1920 Pesikaka, B. H., Shakspear Hut, Gower Street, W.C. 1.

1895 Pickard, Ransom, M.D., 31, East Southernhay, Exeter.

ELECTED

1916 Pike, N., M.D., 10, York Terrace, St. George's Road, Cheltenham.

1910 Pisani, Lt.-Col. L. J., I.M.S. (retired), 49, Wimpole Street, W. 1.

1900 †Pockley, Francis Antill, M.D., 227, Macquarie Street, Sydney, N.S.W.

1903 Pollock, W. B. Inglis, 21, Woodside Place, Charing Cross, Glasgow.

1896 *Pooley, G. H., 304, Glossop Road, Sheffield.

1894 †Pope, R. J., M.D., Box 497, G.P.O., Sydney, N.S.W.

1902 Potter, Bernard E., M.B., 26, Park Crescent, Regent's Park, W. 1.

1903 *Potts, George, Bower Cottage, Tonbridge Road, Maidstone.

1899 Price, Henry J., Maldon, Essex.

1882 Prichard, A. W., 6, Rodney Place, Clifton.

1908 Pridmore, Walter G., Major I.M.S., c/o Grindlay and Co., 54, Parliament Street, S.W. 1.

1903 Pritchard, Eric L., M.D., 70, Fairhazel Gardens, N.W.

1892 Pronger, Charles Ernest, Litchdon, Harrogate.

1909 Prowse, S. Willis, M.D., 706, Union Bank Buildings, Winnipeg, Canada.

1911 Quick, Hamilton E., M.B., "Varfell," 130, Eaton Crescent, Swansea.

1889 Ramsay, A. Maitland, M.D. (*Pres.*), 15, Woodside Place, Glasgow. (C. 1909-11. V.-P. 1915–16.)

1922 Rea, Robert Lindsay, M.D., M.Ch., B.Sc., 125, Harley Street, W. 1.

1899 Read, E. L., Government Medical Officer, 41, Frederick Street, Port of Spain, Trinidad, West Indies.

1921 Renwick, Gordon, Royal Eye Hospital, Oxford Road, Manchester.

1891 Reynolds, Austin Edward, 35, Finsbury Square, E.C. 2.

ELECTED

1913 Riad, M., Kasr el Aino Hospital, Cairo, Egypt.

1897 Richmond, R., M.D., 29, Lingfield Road, Wimbledon, S.W. 19.

1922 Riddell, Brownlow, M.D., 22, Newton Place, Glasgow.

1892 Ridley, Nicholas C., M.B., 27, Horse Fair Street, Leicester.

1921 Roberts, B. H. StClair, Cwm House, Castle Street, Worcester.

1885 *Roberts, Edward, Penywern, Aberystwith, Cardiganshire.

1896 †Roberts, Col. Sir James R., C.I.E., M.B., I.M.S., c/o Messrs. King, King and Co., Bombay.

1891 Robertson-Fullarton, Archibald Louis, M.B., C.M. (Address uncommunicated).

1921 Robinson, F. H., M.B., 1 and 2, Castle Mansions, Eloff Street, Johannesburg, South Africa.

O.M. Rocklitfe, W. C., M.D., Selioke, Hessle, Yorks. (C. 1892-5. V.-P. 1900–3.)

1898 Roe, Arthur Legge, 43, Pryme Street, Hull.

1898 Roll, G. W., M.B., B.C., 7, Upper Wimpole Street, W. 1. (C. 1910-12.)

1890 Rolston, John R., 14, The Crescent, Plymouth.

1891 Roper, Arthur Charles, The Surrobery, Exeter.

1919 Rose-Innes, Arthur, 25, Ashchurch Park Villas, W. 12.

1921 Ross, James Alexander, M.B., Cavendish House, Carlisle.

1882 †Roth, Reuter E., D.S.O., 42, College Street, Hyde Park, Sydney, New South Wales.

1893 *Rowan, John, M.B., 10, Woodside Crescent, Charing Cross, Glasgow.

1899 *Roxburgh, A. B., M.B., 3, Manchester Square, W. 1.

1922 Rubidge, J. L., M.B., C.M., D.P.H., Willowmore, Cape Province, S. Africa.

1895 Russell, J. S. Risien, M.D., 14, Wimpole Street, W. 1. (C. 1900-3. S. 1903-6. C. 1906-9.)

ELECTED

1903 Rutherford, A. Freer, M.B., 19, Abbey Road, Barrow-in-Furness.

1900 †Sager, D. S., M.D., Brantford, Ontario, Canada.

1884 *Sandford, Arthur W., M.D., 13, St. Patrick's Place, Cork. (C. 1896-9. V.-P. 1902-5.)

1918 Santos, Costa, Lisbon, Portugal.

1918 Saunders, P. W., M.B., 63, Wimpole Street, W. 1.

1907 Scott, G. Affleck, M.B., Kingsland House, Ballarat, Victoria.

1921 Seale, Edward A., M.D., B.Ch., Grahamstown, S. Africa.

1918 El Seesy, A. M., Cairo, Ophthalmic Hospital.

1892 Shannon, John Rowlands, M.D., 17, East 38th Street, New York City, U.S.A.

O.M. Sharkey, Sir S. J., M.D., 24a, Portland Place, W. 1. (S. 1885-8. C. 1888-91. V.-P. 1900-2.)

1918 Shaw, David le Baron. (Address uncommunicated.)

1920 Sichel, Alan W., M.D., S.A. Association Chambers, 6, Church Square, Cape Town.

1920 Simpson, William Henry, M.B.

1899 Sinclair, A. H. H., M.B., 3, Charlotte Square, Edinburgh. (C. 1917–1919.)

1891 Sinclair, Walter William, M.B., 3, Arcade Street, Ipswich. (C. 1909-1912.)

1912 Smith, D. Priestley, M.B., 95, Cornwall Street, Birmingham.

1889 Smith, John, M.D., Brycehall, Kirkcaldy.

O.M. Smith, Priestley, M.B., M.Sc., 52, Frederick Road, Edgbaston, Birmingham. (V.-P. 1887-90; 1898-1901; 1907-10. C. 1883-6. *Pres.* 1905-7.)

1903 Smyth, Ernest J., M.B., Sevington, Epsom Road, Guildford.

1911 Snell, H. Cecil, 70, Hanover Street, Glossop Road, Sheffield.

1910 Snellen, Prof. H. Biltstraat, Utrecht, Holland.

1901 Snowball, Thomas, M.B., 83, Bank Parade, Burnley.

ELECTED

1919 Sobhy, N., M.D., c o Ophthalmic Hospitals, Public Health Department, Cairo.

1919 Souter, Wm. Clarke, M.D., 2, Bon Accord Square, Aberdeen.

1889 Spencer, Matthew H., M.D., 92, Oxford Gardens, North Kensington, W. 10.

1889 Spicer, Wm. T. Holmes, M.B., 5, Manchester Square, W. (C. 1900-2. L. 1907-12. V.-P. 1910-12.)

1905 Sprawson, F. C., M.B., 7, Imperial Terrace, Claremont Park, Blackpool.

1895 †Stamberg, A. C., M.B., 5, Windsor Crescent, St. Heliers Jersey, Channel Islands.

1886 Stephenson, Sydney, M.B., 33, Welbeck Street, W. 1. (C. 1898-1901, 1904-7. S. 1901-4. V.-P. 1915–17.)

1896 Stevenson, Edgar, M.D., 39, Rodney Street, Liverpool.

1904 Stevenson, John Simpson, M.B., Royal Exchange Buildings, Cathedral Square, Christchurch, New Zealand.

1920 Stevenson, Thomas, M.D., 40, Rodney Street, Liverpool.

1905 Steward, E. S., 10, Princes' Square, Harrogate.

1913 Stewart, John Barbour, M.B., 17, Woodside Terrace, Glasgow, W.

1893 Stirling, Alexander Williamson, M.D., Empire Buildings, Atalanta, Georgia.

1887 †Stirling, J. W., M.B., 388, Sherbrook Street West, Montreal, Canada.

O.M. Story, J. B., M.B. 6, Merrion Square North, Dublin. (C. 1885-8. V.-P. 1894-7, 1920–22. *Pres.* 1918-20.)

1904 Sykes, Walter, 31, Winckley Square, Preston.

1888 *Sym, William George, M.D., 12, Alva Street, Edinburgh. (V.-P. 1918–21. C. 1906-9.)

1883 †Symons, Mark Johnston, M.D., North Terrace, Adelaide, South Australia.

1907 Tampi, K. R., Trivandrum, South India.

ELECTED

O.M. Tay, Waren, 61, Oakfield Road, West Croydon. (C. 1880-2.)

1903 Taylor, H. H., 36, Brunswick Square, Hove, Brighton.

1899 Taylor, Inglis, M.B., 22a, Cavendish Square, W. 1.

1891 Taylor, James, C.B.E., M.D., 49, Welbeck Street, W. 1. (V.-P. 1910-11. C. 1894-7, 1900-3. S. 1897-1900. T. 1911-1918.)

1889 Taylor, S. J., M.D., 44, Prince of Wales' Road, Norwich. (C. 1904-7.)

1919 Tewfic, M., c/o Ophthalmic Hospitals Siza, Department of Public Health, Cairo.

1900 Thomas, Frank G., M.B., 22, Walter Road, Swansea.

1895 Thompson, A. Hugh, M.D., 36, Weymouth Street, W. 1. (C. 1910-12.)

1895 Thompson, George W., M.B., 80, Harley Street, W. 1.

1895 *Thompson, Robert, M.D., B.S., Bunya Bunya, Wickham Terrace, Brisbane, Queensland.

1911 Thomson, Eric A., M.B., Maison Anton Raad, Beyrout, Syria.

1905 Thomson, H. Wright, D.S.O., M.D., 3, Sandyford Place, Glasgow.

1898 Thomson, W. Ernest, M.D., Viewbank, Bothwell, Lanarkshire.

1919 Thorpe, Reginald, 15, Cavendish Place, W. 1.

1919 Tivy, Cecil B. F., M.B., 12, The Crescent, Plymouth, South Devon.

1883 †Tobin, William, c/o Messrs. John Tobin and Co., Halifax, Nova Scotia, Canada.

1900 Tomlinson, John H., Tudor Lodge, Egham, Surrey.

1904 †Tooke, Frederick T., M.D., 36, Mountain Street, Montreal, Canada.

1895 Toombs, Herbert George, 193, Cromwell Mansions, Cromwell Road, S.W. 7.

1904 Townsend, T. H. D., M.B., 14, St. Patrick's Hill, Cork.

1912 Traquair, H. Moss, M.D. (C.), 16, Manor Place, Edinburgh.

ELECTED

1905 Trench, F. P., M.B., 81, Eaton Terrace, S.W. 1.

1902 †Trilokekar, V. S., 308, Lohar Street, Bombay, India.

1917 Tuckett, Ivor Lloyd, M.D., 2, Trebovir Road, Earls Court, S.W. 5.

O.M. Tweedy, Sir John, LL.D., 100, Harley Street, W. 1. (*Pres.* 1903-5. C. 1884-7. V.-P. 1891-4, 1905-8.)

1898 Tyrrell, F. Astley-Cooper, M.B., 27, Queen Anne Street, W. 1.

1894 Usher, C. H., M.B., 3, Bon Accord Square, Aberdeen. (C. 1909-1912.)

1916 Valentine, John A., M.D., 53, Kent Road, Southsea.

1920 Veitch, A. H., M.B., Birmingham and Midland Eye Hospital, Birmingham.

1913 Viner, Geoffrey, M.D., 27, Queen Anne Street, W. 1.

1922 Vivian, Harold Sugden, M.B., B.S., Chota Dilkuosha, Windmill Hill, Enfield.

1888 Walker, Cyril H., M.B. (V.-P.), 8, Oakfield Road, Clifton, Bristol. (C. 1907-10).

1916 Wallace, William, M.D., 11, Ladbroke Road, W. 11.

1900 Wardale, John D., M.B., Carlton Villa West, Jesmond Road, Newcastle-on-Tyne.

1914 Weeks, John Elmer, M.D., D.Sc., 46, East 57th Street, New York City, U.S.A.

1910 †Weihen, A. Wallace, M.D., 183, Macquarie Street, Sydney, N.S.W.

1887 Wells, Arthur P. L., M.B., 48, Belsize Square, Hampstead, N.W. 3. (C. 1896-9.)

1885 Werner, Louis, M.B., 31, Merrion Square North, Dublin. (C. 1902-5.)

1908 Wharton, John, M.D., 21, St. John Street, Manchester.

O.M. *Wherry, G. E., M.B., Corpus Buildings, Cambridge (C. 1897-1900.)

ELECTED.

1920 White, Harry V., M.C., M.D., 143, Palatine Road, West Didsbury, Manchester.

1895 Whitehead, Arthur Longley, M.B., 31, Park Square, Leeds. (C. 1912–15.)

1920 Whiting, Maurice H., O.B.E., M.B., 9, Welbeck Street, W. 1.

1918 Whittington, Capt. T. H., Oakfield, Carshalton Road, Sutton, Surrey.

O.M. Williams, R., 125, High Street, Bangor, North Wales, (V.-P. 1896-9.)

1922 Williamson-Noble, Frederick A., B.Ch., 27, Harley Street, W. 1.

1920 Wilson, David, M.B., 30, New North Road, Huddersfield.

1920 Wilson, Edwd. Fras., M.B., Glenalt, Omagh, Co. Tyrone, Ireland.

1914 Wilson, James Alexander, M.D., 4, Central Avenue, Cambuslang, Glasgow.

1911 Wilson, S. A. Kinnier, M.D., 14, Harley Street, W. 1. (C. 1918–21. S. 1915-18.)

1921 Wong, Sik To, "Lok-lo-ha," North Road, Hythe, Kent.

1900 Wood, C. G. Russ, 12, St. John's Hill, Shrewsbury.

1889 Wood, David J., M.B., National Mutual Building, Church Square, Cape Town, South Africa.

1903 Wood, Percival, The Grange, Crawley.

1909 Wood, R. Moorsom, 32, Beaumont Street, W. 1.

1899 *Worth, Claud, 36, Harley Street, W. 1.

1910 Worton, A. S., M.D., 7, Manchester Square, W. 1.

1890 *Wray, Chas., Bank Chambers, North End, Croydon.

1898 †Wright, Edward Wm., M.D., 115, Montague Street, Brooklyn, New York.

1908 Wright, F. R. Elliston, M.B., Braunton, North Devon.

ELECTED

1922 Wright, Major Robert E., M.D., B.Ch., D.P.H., I.M.S., Government Ophthalmic Hospital, Egmore, Madras, S. India. (Thomas Cook & Sons, Ludgate Circus.)

1896 Yarr, Col. Sir Michael T., K.C.M.G., C.B., A.M.S., Army and Navy Club, Pall Mall, S.W. 1.

1910 Yearsley, J. Herbert, "Giffnock," 6, Christchurch Road, Bournemouth.

1917 Young, George, M.D., Cambridge Road, Colchester.

1912 Zorab, Arthur, M.B., Winchester House, Hulse Road, Southampton.

RULES.

1. Object of the Society.—The object of the Society is the cultivation and promotion of Ophthalmology.

2. Meetings.—The Society shall hold a meeting each year; the sittings shall be held on two or more consecutive days. On each day there shall be usually two sittings. The date and place of meeting shall be chosen by the Council.

3. Constitution.—The Society shall consist of Ordinary and Honorary members. All medical practitioners whose qualifications are satisfactory to the Council of the Society shall be eligible as Ordinary members. Other Ophthalmological Societies in the British Empire, approved by the Council, may become affiliated.

4. Officers of the Society.—The officers of the Society shall consist of a President, four Vice-Presidents, a Treasurer, two Secretaries and six other members, who together with the President of each affiliated Society or representative accredited by him, shall form the Council and manage the Society's affairs.

5. Election of Members.—Every applicant for admission to the Society shall send to one of the Secretaries a form signed by at least three members, who have personal knowledge of the applicant, or by the President and Secretary of an affiliated Society. Such application shall be submitted to the Council, and if approved the applicant shall forthwith be admitted a member.

6. Honorary Members.—The Council shall have the power of electing as Honorary members men of distinguished eminence in Ophthalmology, or in the sciences bearing upon it.

7. Subscriptions. — The Annual Subscription shall be £1 11*s*. 6*d*. payable in the month of October. No member whose subscription is in arrear shall be entitled to attend the meeting. Any member whose subscription is twelve months in arrear shall cease to be a member of the Society.

8. Election of Officers.—The Officers of the Society shall be elected yearly by Ballot at a Business sitting held during the meeting. A balloting list of the names recommended by the Council for election shall be posted to each member two weeks previously. At the Business sitting twelve shall form a quorum.

9. Duration of Office.—The President shall not hold office for a period of more than two consecutive years. No other member of Council, except the Treasurer, shall hold the same office for more than three consecutive years.

10. President and Vice-Presidents.—The President shall regulate all the proceedings of the Society and Council, state and put questions, interpret the application of the laws, and decide any doubtful points. He shall check irregularities and enforce the observance of the laws. He shall sign the minutes of General and Council meetings. In the absence of the President one of the Vice-Presidents, the Treasurer, or some other member chosen by the meeting, shall perform his duties.

11. Secretaries.—The Secretaries shall manage all correspondence, and shall keep the Minutes of the Society and Council. They shall notify to new members their election. They shall arrange with the President the order of proceedings at the meetings. They shall have charge of and shall keep a register of all papers communicated, and shall be the Editors of the 'Transactions.'

12. Treasurer.—The Treasurer shall receive all moneys due to the Society, and make all payments ordered by the Council, keeping an account of all such receipts and payments. The accounts shall be audited once a year and presented to the Society at the Annual Business Meeting. Two members of the Society shall be nominated by the President to audit the Treasurer's accounts.

13. Meetings of Council.—The Council shall meet as frequently as necessary during the Annual Meeting and at other times if specially convened. Five shall form a quorum. The President shall have a casting vote in addition to his ordinary vote. The Council shall decide upon all questions relating to the reception of communications, and shall have power to submit any paper to referees.

14. Vacancies of Offices.—The Council shall have the power of filling up any vacancies which may occur in any of the offices of the Society between one Annual Meeting and the next.

15. 'Transactions.'—The Society shall publish a yearly volume of 'Transactions' as soon as possible after the Annual Meeting (*vide* Bye Law 1 concerning communications). There shall be an Editorial Committee consisting of the President, the Secretaries and two other members of Council, together with the Secretaries of affiliated Societies. A copy of the 'Transactions' shall be sent to every member whose subscription has been duly paid; and to each Honorary member.

16. Publication of Papers and Discussion.—No paper shall be accepted if it has been published elsewhere. No paper shall be read before the Society unless a copy of it has been sent to the Secretaries at least four weeks before the meeting, together with an abstract suitable for immediate publication in the journals. When possible, notice should be given relating to card cases, and a short abstract of all papers to be read before the Society shall be distributed to all members before the meeting. No report of the meetings of the Society may be published by members or others without the sanction of the Council.

17. Notice of Meeting.—Three months before the date of the Annual Meeting a preliminary notice of the meeting shall be sent to every member.

18. Visitors.—Any member of the medical profession may attend a meeting of the Society (except the Annual Business Meeting) on being introduced by a member of the Society.

The Library has now been transferred to the premises of the Royal Society of Medicine, 1, Wimpole Street.

LIBRARY RULES.

1. The Library shall be open at the same hours as that of the Royal Society of Medicine, viz. from 11 a.m. to 6.30 p.m. daily, except Sundays.

2. Members will be entitled to read the books at 1, Wimpole Street, between these hours, or to take them out on signing a receipt provided for that purpose.

3. A large number of periodicals will be accessible to members in the Library. The current number may not be taken out of the Library.

4. A book must be returned at the expiration of a fortnight if wanted by any other member. The Librarian will in such a case write to the member in whose name the book was taken out.

5. If the book be not returned within four days of such notice a fine of 6d. will be charged for each day that the book is retained beyond such days of grace.

6. Instruments and drawings cannot be taken out of the Library except by special permission.

7. A member taking out a book will be held responsible for its being returned in good condition.

8. Members of the Ophthalmological Society who are also Members of the Section of Ophthalmology of the Royal Society of Medicine can have the full use of the Fellows' Library on payment of an additional guinea instead of the usual subscription of two guineas.

ADAMS FROST COLLECTION OF LANTERN SLIDES.

1. The slides shall be in the custody of the Librarian.

2. They may be lent, for the purpose of teaching, to teachers in any recognised Medical School on application to the Librarian.

3. The regulations under which the slides may be borrowed, together with a list of the slides, shall be printed (*a*) in the *Transactions*; (*b*) in the Catalogue of the Library; (*c*) on leaflets which may be sent to enquirers.

4. Other slides may be added to the collection, but the numbering (in so far as it coincides with that of the figures in Frost's *Fundus Oculi*) shall be retained.

5. The following are the regulations under which the slides may be borrowed:

Regulations for the Loan of Lantern Slides.

1. Not more than 20 slides may be borrowed at one time.

2. Not less than three days' notice of the wish to borrow slides shall be given, and a list giving the numbers of the slides required shall be sent to the Librarian.

3. The borrower shall sign a receipt for the slides, which will be given back to him on their return.

4. The slides must be returned within three days, no other slides can be lent to the same borrower till this has been done.

5. Any slides broken, lost, or damaged shall be replaced or repaired at the expense of the borrower.

THE BOWMAN LECTURE.

Resolution of Council, September 18th, 1883.

"That in recognition of Mr. Bowman's distinguished scientific position in ophthalmology and other branches of Medicine, and in commemoration of his valuable services to the Ophthalmological Society, of which he was the first President, the Council shall each year, or periodically, nominate some person to deliver a lecture before the Society, to be called 'The Bowman Lecture,' which shall consist of a critical *résumé* of recent advances in ophthalmology or in such subject or subjects as the Council shall select, or of any original investigation, and shall be delivered at a special meeting of the Society held for the purpose, at which no other business shall be transacted."

EDWARD NETTLESHIP PRIZE.

On the occasion of Mr. Nettleship's retirement from practice in 1901 a fund was inaugurated by his friends and pupils, with the object of founding an Edward Nettleship Prize for the Encouragement of Scientific Ophthalmic Work, and at a meeting of the General Committee of the above fund on July 2nd, 1902, the following resolutions were adopted :

1. That the prize should be a Gold Medal for the encouragement of scientific ophthalmic work.

2. That the Council of the Ophthalmological Society of the United Kingdom be asked to undertake the custody of the Prize and Fund, and to appoint Trustees.

3. The award of the Medal shall be entrusted to three members of the Ophthalmological Society appointed by the Council, such members to be changed after each award.

4. That the Medal shall be awarded at such intervals as shall be determined upon.

5. That only British subjects be eligible to receive this Prize.

6. That, subject to the discretion of the said Council, the Prize shall be awarded—

(*a*) For the most valuable contribution to Ophthalmology during the three years immediately preceding or since the last award, or

(*b*) For the best work done on any subject previously selected and announced by the Council of the Ophthalmological Society.

7. That in the event of no work being found of sufficient merit an award shall not be made.

Resolution of Council, January 25th, 1906.

"It shall be lawful for the Trustees, with the consent of the Council of the Society, to expend any surplus arising from the non-award of the Medal in any one year, or accumulated during several years, in the purchase of works for the Library of the said Society, such works to be inscribed 'Purchased by the Nettleship Prize Fund'; or, with the consent of the said Council, to expend such surplus in any manner calculated to promote the objects of the Society as defined in the Rules."

N.B.—Additional trustees, in accordance with the terms of the Trust Deed, have to be appointed by some person nominated by the Council.

BYE-LAWS CONCERNING COMMUNICATIONS.

1. The 'Transactions' shall consist of such communications made to the Society, or to affiliated Societies, by or through

members, as may be deemed by the Council suitable for publication. Also of discussions of importance or interest arising out of such communications.

2. No communication to the Society shall occupy more than twenty minutes, and in the subsequent discussion of it no member shall speak more than once, or for more than ten minutes, without the special permission of the Chairman.

3. All communications accepted by the Society become the property of the Society.

4. Communications are admissible which may have been read elsewhere, provided they have not been published.

5. The cost of illustrations shall be borne by the Society so far as, in the opinion of the Council, is consistent with the state of its funds.

6. Reprints of papers may be obtained by authors at their own expense, by arrangement with the printer.

REGULATIONS CONCERNING THE EXHIBITION OF PATIENTS AND OF PATHOLOGICAL SPECIMENS BY CARD.

A. A card, provided by the Society, must be placed conveniently near the patient (unless it is undesirable that it should be read by the patient or friends), and on it must be clearly written an account of the case, *comprising all the particulars intended for publication*. The title only of the case will be announced by the President to the meeting, but the Exhibitor (or his representative) must be present at the meeting, and be willing to read the case and furnish additional details if called upon to do so; the length of such oral communications not to exceed five minutes.

B. Pathological Specimens may, at the discretion of the Exhibitor, be shown by card, and will then be subject to the above regulations. It is particularly to be noted that the description on the card must *comprise all the particulars intended for publication.*

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OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM.

I. OPENING OF CONGRESS.

The Annual Congress was held on May 11th to 13th, 1922, at the House of the Royal Society of Medicine and St. Bartholomew's Hospital. Mr. J. Herbert Fisher, M.B., F.R.C.S., President, in the Chair.

Before beginning the business of the Congress, the Edward Nettleship Prize was presented to Dr. H. Moss Traquair by the President for his work on "Bitemporal Hemianopsia."

REPORTS.

II. DISCUSSION ON INDUSTRIAL DISEASES OF THE EYE

(Excluding Accidents, Miners' Nystagmus, and Glass-Blowers' Cataract).

Opening Paper.

By Dr. T. M. Legge, C.B.E.,
H.M. Medical Inspector of Factories.

Mr. President, Ladies and Gentlemen,—It was only under considerable pressure that I was induced to address you in opening the discussion to-day, because I do not want to sail under false colours, and it is on the administrative side, and not the clinical side, that I wish to be judged.

Excluding accidents, glass-blowers' cataract and nystagmus, the diseases of the eye due to occupation are few—acute conjunctivitis from light from the arc formed in electric welding, such light having excessive ultra-violet rays, from certain irritating gases like hydrogen sulphide, from dust of certain kinds of wood, such as satin wood, teak wood, and East Indian boxwood, and from certain alkaloids and chemical substances: chronic conjunctivitis and chronic inflammation or staining of the cornea from tar and pitch, and mercury and silver fulminate: optic neuritis from lead, nitro and amido derivatives of benzene, carbon bisulphide and tobacco: and lastly albuminuric retinitis as a sequela in the chronic nephritis set up by lead.

I remember saving a chemist in a works, suffering from an intractable conjunctivitis in one eye, fruitless journeys by to town by noticing a slight smell of sulphuretted hydrogen in connection with a chamber, to a peep-hole in which he applied the affected eye periodically. It was the stream of sulphuretted hydrogen he thus received which accounted for

the condition. Similarly, in the early days of the manufacture of sesquisulphide of phosphorus to replace yellow phosphorus in the making of lucifer matches, the life of the manager of the only factory where this is made was rendered miserable by conjunctivitis set up by the small amount of sulphuretted hydrogen given off on the material coming into contact with the moisture of the air. This was only prevented by placing the manufacturing process and storage bins under a negative pressure by means of exhaust ventilation.

I wish to confine my remarks to the potential danger which still exists from amblyopia, and to show you how it is kept in check. The following figures show you the way in which lead poisoning has diminished by more than one half since the year 1900 :

Industry.	Average, 1900–04.	Average, 1905–09.	Average, 1910–14.	Average,* 1915–19.
Smelting of metals	37 (2)	45 (2)	40 (4)	34 (2)
Brass	8	7	7	1
Sheet lead and lead piping	13	9	7	2
Plumbing and soldering	20 (1)	23 (2)	32 (2)	19 (1)
Printing	18 (1)	22 (2)	29 (2)	13 (2)
File cutting	32 (3)	11	13 (1)	3 (1)
Tinning	10	18	13	3
White lead	183 (4)	76 (3)	31 (1)	17
Red lead	13	9	8	11
Pottery	119 (5)	94 (7)	67 (10)	19 (6)
Litho transfer-making	5	5	1	—
Glass cutting and polishing	6 (1)	3 (1)	2 (1)	—
Enamelling	6	4	12	2
Electric accumulators	32	25 (1)	36	40 (1)
Paints and colours	46 (1)	39 (1)	20	12
Coach-building	64 (4)	75 (4)	77 (5)	23 (3)
Ship-building	29 (1)	24 (1)	31 (3)	16 (2)
Paint used in other industries	46 (2)	45 (2)	49 (2)	16 (1)
Other industries	66 (2)	65 (2)	73 (3)	48 (2)
Total	753 (27)	599 (28)	548 (34)	279 (21)

The principal figures relate to cases, the figures in parentheses to deaths. Fatal cases not reported in previous years are included as both cases and deaths.

* The diminution in the figures for the period 1915–19 is largely the result of the partial stoppage of many lead industries as a consequence of the war.

The diminution, you will notice, is confined mainly to the cases occurring in white lead, in china and earthenware manufacture, and in paints and colours, and is not shown in the industries where paint is used—at any rate down to the year 1914, as, for example, in coach painting, ship painting, or paint used in other industries. If you understand the precise meaning of that you will understand how the risk of poisoning is controlled.

These further figures for the years 1905-1919 show the severe cases that have been included first under the general head of "encephalopathy" and again under "optic neuritis":

Encephalopathy.

Symptom.	Total, 1905-09.	Total, 1910-14.	Total, 1915-19.
Epilepsy and epileptiform attacks	64	39	8
Mental defect	13	16	3
Optic neuritis	20	5	6
Total	97	60	17

You will notice that there has been the same happy diminution in them that there has been for the general run of the cases. These results have been largely accomplished by the advance made in locally applied exhaust ventilation by means of fans to remove dust and fume at the point at which it is produced, and so prevent its inhalation by the workers. It applies not only to lead, but also to illness from carbon bisulphide, and also in some measure to nitro and amido-derivatives of benzene. If you stop the dust and fumes, you will stop the poisoning. There are other ways besides exhaust ventilation by which the same object is achieved, as, for instance, the use of mechanical means reducing hand labour, the conversion of white lead into paint without first drying it into a powder, which is now the usual practice, the substitution of low solubility glazes for raw lead glazes in pottery, and so on.

When I came into the Factory Department in 1898, it was quite in the ordinary course of inspection to have to follow up a case of blindness due to lead. In 1898 Mr. Folker, then Ophthalmic Physician to the North Staffordshire Infirmary, published notes on eight recent cases of amblyopia where causes other than lead could be excluded. Sir Thomas Oliver dealt with the same aspect in his Goulstonian Lectures of 1891 with reference to the white lead works in Newcastle. It is difficult to say precisely how far the optic nerve is affected in cases of lead poisoning under conditions controlled by exhaust ventilation. A general practitioner in the Potteries in 1910 stated, as a result of his observation of 410 cases, that comparing men and women he had found the following figures :

Comparative Percentage.

	Males. Per cent.	Females. Per cent.
Paralysis	57·0	30·0
Totally blind	2·3	7·7
Partially blind	3·5	10·2
Eyes affected	10·0	14·0
Gastric disturbance	77·6	69·8
Epilepsy	17·6	17·0
Encephalopathy	15·0	34·9

Unfortunately I find that writers on industrial disease sometimes allow their enthusiasm to run away with their discretion, and I personally consider these figures as exaggerated, although I am just as ready to admit that the figures I give you, which are based on reports of notifications received and carefully scrutinised, err on the other side and give *couleur de rose*.

How easily a disturbance of the normal course of manufacture allowing greater exposure gives rise to a sudden outbreak as in 1914 is illustrated by an instance just prior to the war, when no less than twenty-eight cases of illness, including four of amblyopia, occurred in a factory manu-

facturing explosives containing about 10 to 12 per cent. of di-nitro-benzene. As long ago as 1894, Snell published in the *British Medical Journal* an account of five cases of optic neuritis due to this cause. A great demand for the explosive arose, and to meet this extra rush of orders, first overtime was worked, next a night shift was put on, and as the summer progressed and the heat as well as the volatility of the substance increased, there were more men on at night time than during the day. When the shifts were reduced to four hours the conditions at once improved.

Dr. Holmes, Ophthalmic Surgeon to the Royal Infirmary, Wigan, stated: "I have never before seen di-nitro-benzol cases with such extreme contraction of the fields of vision as in two of the cases, and find it hard to know what is the true significance of this. Usually contraction of the field of vision means atrophy or hysteria. I did not see any other symptoms of hysteria or neurasthenia in these men, and do not think this is the explanation, and in none of the cases did the field for the movements of the hand appear to be greatly limited; therefore I hope there is no real atrophy. However, this contraction with marked pallor must be considered a very serious symptom, and I think all of the men should be absolutely removed from the possibility of coming in contact with the poison, especially as there seems to have been no improvement during the three months."

During the war T.N.T., which accounted for so much illness from dermatitis and toxic jaundice, did not, as does di-nitro-benzene, affect the optic nerve. Two cases only were brought to my notice, and in these I had the advantage of reports by an ophthalmic surgeon—Dr. J. V. Paterson, of Edinburgh—who considered the weight of evidence was in favour of tobacco rather than T.N.T.

Last year my colleague, Dr. Middleton, followed up a case of a woman employed for twenty-five years in the leaf room at a tobacco factory in Edinburgh. About nine months previously she began to feel her vision defective. Her case was diagnosed by Dr. W. G. Sym, Ophthalmic Surgeon to the Edinburgh Royal Infirmary, as tobacco amblyopia, due

to finely divided tobacco dust passed through a fine-meshed hand-sieve, to which she had been exposed. This is the only case that has been brought to my notice in this country.

There are two points on which I should like the opinion of the meeting, and put forward partly also as subject for discussion. There are certain diseases which I look upon as the most strictly occupational of any, as they are due to constant stress and strain in which physiological strain has passed the border-line and become pathological. As Prof. Kent has said, only when the breaking down is exaggerated and the process of repair fails to keep pace with the process of destruction does fatigue of an injurious degree supervene. Thus, in persons of neurotic temperament, the central nervous system may fail to co-ordinate the muscular movements, as in writers' and telegraphists' cramp. Similarly, as the results of strain, the psycho-neurosis of miners' nystagmus is brought about by deficient light. How far can the increase of the long axis of the eyeball constituting myopia be regarded as an occupational disease? With all the talk there has been about industrial fatigue, apart from the subject of nystagmus, it is remarkable how little has been done on the subject of visual fatigue. It must have come prominently before all who are here.

Is the fine work, as in the compositor, responsible for the number who are found with myopia, or does the work merely call attention to the errors of refraction that were there already? And bearing on this there is another point that I throw out for discussion: Why is it that with the constant strain to which the eyes of a person with uncorrected errors of refraction is subjected in the course of his daily work, he does not develop nystagmus? I cannot understand how eye-strain in semi-darkness can cause a peculiar train of symptoms, and eye-strain, due to uncorrected errors of refraction, should not.

Opening Paper.

By A. Bernard Cridland.

Mr. President and Gentlemen.—May I first express my thanks to you and the Council of the Society for the compliment paid me in asking me to assist in opening this discussion.

With the exclusion of miners' nystagmus, glass-blowers' cataract and accidents to the eye in general, and in addition to the diseases already referred to by Dr. Legge, we are at liberty to consider two others, namely, the occurrence of cataract amongst workers at furnaces other than those used in the glass trade, and, secondly, ulceration of the corneal surface of the eye from tar, pitch, mineral oil or paraffin, or any other compound product or residue of these substances.

Finally, we may consider any other forms of employment which may possibly be responsible for ocular diseases but which can be regarded at the present time as suspects only.

I would first draw your attention to the occurrence of cataract amongst furnace-workers, which, as you are all aware, now entitles these men to a small compensation under the Workmen's Compensation Act.

It is the latest addition to the Home Office List and came into force on December 1st, 1921.

It is described as "cataract caused by exposure to rays from molten or red-hot metal," and the circumstances in which the disease may arise are "processes normally involving such exposure in the manufacture of iron or steel."

It had long been suspected that lental opacities would be found amongst those whose work, beside that of the glass trade, exposed them to great heat and light: and although some investigation was made in 1909, no positive evidence was forthcoming until a case of the kind was shown in 1915 at a meeting of the Ophthalmic Section of the Royal Society of Medicine.

A man æt. 50 years, who had worked as a puddler for thirty years and who had never been in a glass-furnace, showed opacities in both lenses of the well-known saucer-

shaped type and similar in every way to that found in glass-blowers.

Shortly afterwards St. Clair Roberts, searching amongst chain-makers, found evidence of lental opacities which could only be due to the nature of the employment. These opacities, however, differed somewhat from the saucer-shaped type, for the majority of the cases presented a dense posterior polar or posterior cortical opacity, with an absence of the surrounding semi-opaque ring. In addition, a small anterior polar opacity was not infrequently present, and the type in general was not unlike that found in the earlier stages of the glass-blowers' cataract.

Later still, Healy, in an excellent and exhaustive paper, published in the *British Journal of Ophthalmology* the results of his findings in mill-men, *i. e.* men who have the handling of red-hot bars of steel which are rolled out into plates. As with puddlers and chain-makers, he found the posterior polar and cortical opacities, but in a comparatively small number of cases.

In a much greater proportion there was present what he described as "a striated cortical wedge-shaped opacity, apex upwards, growing in the lower cortex or downwards and slightly inwards," whilst sometimes this opacity was broom-like in appearance though still of the same shape.

The actual figures are: In 209 cases examined 105 examples of the wedge-shaped variety alone, and of the posterior cortical 26 cases, whilst 20 showed a combination of the two varieties, and in four a nuclear opacity alone was present.

It would seem, then, that so far we can distinguish three broad types of lental opacity in furnace-workers:

(1) The saucer-shaped variety seen in puddlers and mill-men, and similar in every respect to that found in glass-blowers.

(2) The posterior polar or blot-like opacity as exemplified in chain-workers.

(3) The much more common wedge-shaped opacity seen in mill-men and possibly other furnace-workers.

The first two types, which appear to be by no means common, are well recognised, and I do not think any doubt can be

raised but that they are directly due to the nature of the employment. From the situation of the opacities in the lens, and from the researches which have been made upon the effects of heat-rays upon this structure, the evidence seems to be complete, and we shall be correct in saying that these two types fall within the wording of the Home Office list.

In the third type we have, however, an opacity which occurs very much more commonly—roughly in 50 per cent. of all the furnace-workers. The situation of the opacity in the lens makes its ætiology difficult to explain if we are to take heat-rays as the direct cause, and we have to remember that it is not an uncommon form amongst those who have never been amongst furnaces—at least for any prolonged period of time. At the same time we are faced with the evidence brought forward that opacities in the lens are frequently seen in these mill-men, and at an age as early as round about 35.

Incidence of Cataract at Various Ages in Mill-men and in Ordinary Refraction Cases.

	Age.							
	35–40.	41–45.	46–50.	51–55.	56–60.	61–65.	66 and over.	All ages.
(1) In mill-men (Healy):								
Number of men examined	74	67	65	61	53	29	—	
Number found to be suffering from lenticular opacities	13	12	22	38	34	20	—	—
Percentage	17·5	18	34	62	64	69	—	—
(2) In ordinary refraction cases:								
Number of patients examined, 1000	116	176	222	168	103	74	141	1000
Number suffering from lenticular opacities	18	8	19	25	23	33	72	198
Percentage	15·5	4·5	8·5	14·8	22·3	44·5	51	19·8
Number of men examined, 351	34	64	82	62	35	29	45	351
Number suffering from lenticular opacities	4	3	3	6	5	12	24	57
Percentage	11·7	4·6	3·6	9·6	14	41	53	15·8
Number of women examined, 649	82	112	140	106	68	45	96	649
Number suffering from lenticular opacities	14	5	16	19	18	21	48	141
Percentage	17	4·4	11·4	17·9	26·4	46·6	50	21·9

I would here like to show Healy's table, which gives the incidence of cataract amongst mill-men at the various ages from 35 onwards. The numbers of cases examined are indicated with the number in which cataract was found and with the percentages worked out.

For comparison I have taker 1000 consecutive cases from my private case-books, consisting only of people who presented themselves for refraction error, and excluding inflammatory cases and diseases generally. The percentages have been worked out for men and women together, and for men and women separately.

Comparing these results with those amongst the mill-men, it is to be noted that from 35 to 40 there is a small difference only in the percentage, but that there is a much greater difference in the next quinquennium and for each subsequent quinquennium, although diminishing as the last one is approached.

We cannot therefore but acknowledge that cataract, in mill-men, at least, is more common than in the rest of the population, although the type of opacity much the most commonly found is one which is also found amongst the rest of the population.

As before said, however, the situation in the lens of the wedge-shaped opacity, together with the fact that it is so different from the posterior cortical one, makes it difficult to say that the cause of this type is the direct effect of the heat-rays, and it would seem as though we must look for some other factor to explain its occurrence.

Such may be found in the men themselves, their habits formed by reason of the nature of their work, and the effect of the work upon their metabolism in general. Perhaps a close study of these men may reveal an aetiological factor in the evolution of cataract in general hitherto unknown. We can at any rate regard furnace work as being calculated to make men grow old before their time.

I desire specially to draw the attention of the meeting to the consideration of this third type of opacity, for from an economic point of view there is more in it than meets the eye.

If we regard any type of lental opacity (excluding, of course, congenital forms) which may occur in furnace-workers as being industrial in origin, it is quite certain that we shall be including a certain number which are not industrial, and so we shall be inflicting some injustice on the employer when the time comes that the worker is incapacitated from work.

On the other hand, if we do not regard the majority of these opacities as industrial in origin, we shall be doing an injustice to the men. Can we, then, say that the wedge-shaped opacity is a pathognomonic form of heat-cataract in the same sense that we can regard the posterior cortical forms? If a definite expression of opinion upon this point can be made by the meeting, the path in the future will be smoothed for the certifying surgeon and the specialist medical referee, especially for the latter, as his decision is final and from it there is no appeal.

Another point in reference to this disease, and one which refers also to glass-blowers' cataract, is the question of the amount of compensation to which these men become entitled. The regulation states that compensation shall be paid for a period not longer than six months in all, not for more than four months unless he has undergone an operation for cataract. This presupposes that the man can earn as much with aphakic eyes as he could before his cataract prevented him from working.

I have no hesitation in raising this point, as it involves the scientific expression of opinion as to what a man with aphakia and wearing suitable glasses can accomplish in the conditions concerned.

The view the county court judge takes in his mind upon compensation cases in general is: "What could the man earn before the accident? What can he earn now?" and, after all, an industrial disease is but an accident so oft repeated that it gives rise to a permanent injury. In many other walks of life, no doubt, an aphakic man can carry on just as well as before his lenses were removed.

I recently learned that a gentleman whose lenses I extracted at the age of 58 and 59 respectively has since learnt to drive

a Ford car. His resulting vision was moderate, namely, $\frac{6}{12}$ and $\frac{6}{9}$, but I understand that he drives in traffic with the greatest confidence in himself, although I am not sure that his passengers share the same feelings. Be that as it may, my experience of furnace-workers after operation is that they are unable to earn the same wages as before. Robinson, with his wide experience, states that "after removal of the opaque lens the sight is usually good, although the finisher is rarely able to resume his old employment, owing to loss of accommodation."

On the whole, I think the aphakic man at a furnace and in a mill is under a considerable disadvantage, and for this reason the compensation is insufficient. I suggest that this is a point for discussion.

The consideration of the visual results after operation raises the question as to whether the eye is injured in any other way by the exposure to heat-rays.

In the comparatively few cases amongst puddlers on whom I have operated the vision has never been normal, although from a surgical point of view the result has been all that could be desired, namely a perfectly clear pupil with a good view of the fundus obtainable. I have not been able to detect fundus changes that might not be explained by other causes; nevertheless I am rather of the opinion that the retina does suffer from prolonged exposure to heat-rays.

What evidence can we bring forward bearing upon this?

Fortunately, I am in the position of being able to refer to a valuable paper which has not yet been published, but which certainly supports my view.

It is one by Dr. James Fison, of Harrogate, and I here desire to cordially thank him for permitting me to make use of his work before he has himself brought it publicly forward. His paper is entitled "Stokers' Amblyopia," and formed the subject of his thesis for the Cambridge M.D. in 1918.

Whilst serving in the Royal Navy his attention was drawn to the fact that out of 371 consecutive cases of stoke-hold ratings with defective vision, upwards of forty were found to

have a visual acuity of less than $\frac{6}{18}$ in either eye after correction with glasses, whilst no opacity of the media or disease of the fundi could be discovered sufficiently to account for this poor vision, and in none of the cases could this condition be regarded as due to extra-ocular disease, *e. g.* retrobulbar neuritis, disease of the optic tracts or toxic amblyopia.

In making these observations all cases in which there was a possibility of malingering were excluded, but these were few.

The author states—"It must be pointed out that normal visual acuity, *i. e.* $\frac{6}{6}$, without correcting lenses is not required of stokers. The King's Regulations require an acuity of $\frac{6}{8}$ in each eye on admission, though during the war $\frac{6}{12}$ has been considered sufficient. This standard has been determined as indicating an ability to read the water and steam-pressure gauges without having to go close to them.

"Allusion is made to this subject here in case effective criticism should be brought to bear upon the observations forming the chief *raison d'être* for this thesis, on the ground that there was nothing to prove that the vision of these stokers in whom objective examination failed to reveal the cause of amblyopia was not their normal vision, and that they were deceiving themselves in thinking that there had been any deterioration. It is, however, certain that all the stokers had passed a test upon entry requiring an acuity of at least $\frac{6}{12}$ in each eye without glasses, while another examination had been passed before promotion to chief stoker and stoker petty officer. It is improbable that any man would be able to get to the position of stoker petty officer if his vision were less than $\frac{6}{12}$ (only one such case has been met with). An equal degree of improbability does not apply to the others, and it is possible that in some of the cases the recruiting examiners had been deliberately deceived by the candidates.

"The possibility of the cases of stokers' amblyopia being due to some mild retrobulbar disease was, of course, carefully considered, and, so far as present knowledge will permit, excluded.

"In all the cases examined the visual fields were normal for white and no colour scotomata were found. Cases were excluded in which headache was a symptom.

"The conclusion was drawn that the amblyopia in these patients was due to the condition of their work, and it seemed likely that the fundi were effected by radiant energy waves from the furnaces."

The paper is further given up to a review of the work already done by various authors upon the effects of heat and light upon the eye, and Fison concludes that there seems no doubt but that heat-rays of a certain length can and do pass through to the retina, and are accountable for the reduction of vision in these men.

All the cases were examined in a most careful and complete manner, and the author is to be congratulated on a valuable piece of work, which I hope will soon be published. I again desire to tender him my thanks for allowing me to make use of it on this occasion.

One concludes, then, that it is more than a possibility that the retina participates in the disturbance set up by heat-rays as well as the lens, and it would be interesting to know why in one case one structure, namely, the retina, should suffer and the lens escape.

With this finding in naval stokers one naturally turns one's thoughts to stokers in other work, and especially to firemen on railway locomotives.

The question of vision is of greater importance here, for most firemen are candidates for driving—an occupation in which perfect visual acuity is a *sine quâ non*. Doubtless you are all aware that in the regulations prescribed for a fireman it is one of his duties to observe signals when not engaged in stoking. In the cases of firemen that I have had the opportunity of examining and questioning, I find that they all for a shorter or longer period have difficulty in adapting their eyes to observe signals after gazing into the fire: no doubt this is due to the effect of glare, and whether the prolonged exposure to heat from the furnace has any permanent effect upon their eyes I do not know.

but I suggest that this is a "suspect" occupation, and that in view of the responsible nature of their work investigations would not be without value.

With regard to the other disease in the Home Office schedule, namely, ulceration of the corneal surface of the eye from tar, pitch, etc., I have little to say.

I do not see many cases, but those I do usually come from the well-known firm of Tarmac Limited. In these the cornea is actually injured by pieces of slag, etc., impregnated with tar; ulceration follows, against which treatment seems to be of little or no avail. One cannot but deplore the length of time during which inflammatory changes persist or the permanent damage to the eye when these have subsided.

In conclusion, mention may be made of one occupation that seems to me to be not beyond suspicion; it is that of workers with bright tin.

During the whole day these men are exposed to reflection of light from a very bright surface, and complaints are frequently made by them of the trying nature of their work—from an ocular point of view. I have so far not discovered any lesion which may be attributable to the work. I have found three cases of chronic glaucoma occurring in the small total of those employed in my district, but I would hesitate to suggest any connection between the two. I merely mention this occupation as being a possible suspect, and that perhaps other observers may have something to say about it.

Opening Paper.

Visual acuity and vocational selection.

By Prof. Edgar Collis.

There are signs that more advanced employers of labour are coming to recognise the disadvantage which accrues from the worker and his work being ill-mated: output is lessened: earnings are smaller; and discontent is generated by a worker seeing his fellow doing more and earning more than he can

himself. This lack of agreement between the man and his process is also a potent factor in determining that heavy financial loss known as labour turnover, namely, the tendency for workers to drift from one place of employment to another trying to find congenial occupation.

The science of fitting candidates with suitable work is known as vocational selection; but behind it lies an earlier science, the science of vocational guidance. This guidance should lie with teachers and juvenile advisory committees, who, after considering the ascertained capacity of each adolescent, should guide him to that walk of life wherein such capacity is needed and has scope.

No faculty has greater influence upon capacity than that of vision. All our actions and the majority of our thoughts are originated or guided by visual impulses reaching our central consciousness. As we see, so do we react; even the senses of smell and taste are closely associated with that of sight. The fancy may even be indulged in that artistic appreciations must be dependent to no small extent upon visual acuity and errors of refraction. Thus a carping ophthalmic critic may see in pre-Raphaelite pictures with their minute detail and lack of perspective the productions of a school of myopes; and in impressionist pictures with their lack of detail and blurred outlines, of a school of presbyopes; and point to how the works of Millais tend, as he grew older and his vision altered, to pass from the myopic school to the presbyopic; while, if he gives his imagination full play, he may see in certain up-to-date canvasses of the cuboidal school clear indications of compound astigmatism; and possibly regret that the science of vocational guidance does not guard the entry to the studio. Similarly it is hard to believe that the colour appreciation of an Orchardson is the same as that of an Alma Tadema, or a Yeend King. And the disbeliever will find support in the researches of Edridge-Green, who finds every variety of colour vision from persons who can distinguish seven colours to those who can distinguish only two. When attention is directed to the use of vision in occupation, two aspects of the case are presented: there is

the effect of visual acuity upon industrial efficiency, and the effect of occupations upon visual acuity. Only by reviewing these two aspects of the subject can the need for vocational selection be fully grasped.

Effect of Visual Acuity Upon Industry.

A cursory consideration of occupations establishes how they make a call for accurate vision. In the interest of public safety, the need for the engine driver and the seaman to possess a certain standard of colour vision is recognised by rules and regulations. In the same interest surely there is need, in these days of rapid road traffic, for the visual acuity of motor drivers to be tested. Day after day the papers record accidents in which the same drivers have been involved for the second, third or fourth time. I know of one case where a driver has killed two persons and been involved in a third bad smash. The magistrates are usually content to admonish, to warn, to fine and endorse licences, and then to leave the offender loose as a danger to himself and an unsuspecting public. On no occasion do we hear that the driver has been referred to have his eyesight tested. One instance comes to mind of a chauffeur considered by his employer a marvel because of his capacity for extricating himself from difficult positions. He certainly possessed wonderful control over his engine. One day when being driven by him, I saw a child start across the road; he did not notice what was taking place for another second or so. Then followed a marvel of driving which resulted in the child only being knocked down instead of being killed, and the ruin of two tyres. Had I been at the wheel no accident would have occurred. A few days later I found he could not read the name of a street plainly visible, and his services were dispensed with. I wonder how many ophthalmic surgeons take the precaution, fundamental to the safety of themselves and those to whom they generously give lifts, of testing the visual acuity of their chauffeurs.

Turn now to the question of industrial accidents. Here the conclusion has been come to that about 85 per cent. of

all accidents are due, not to the absence of guards and safety appliances, but "to such causes as negligence, carelessness, want of thought, and perhaps, more than all, to lack of proper appreciation of the danger." At the same time Greenwood has pointed to the existence of varying individual susceptibility to accident; but no one yet has attempted to establish a connection between it and imperfect vision. Nevertheless the relation between deficient illumination and accidents has been pointed out by Wilson and Paterson in this country and by Simpson in the United States; the latter authority estimated that out of 91,000 accidents, 23·8 per cent. were due to imperfect illumination, which, after all, is only an environmental method of diminishing visual acuity. The probability cannot be passed over that innate imperfect visual acuity which certainly exists may be an important factor in determining individual susceptibility to accident. Indeed, statistical proof is hardly needed to establish the case that muscular action is less accurate and more liable to error where vision is imperfect.

Visual acuity must also affect industrial efficiency, but, so far as I am aware, no one has ever examined the best and worst workers at different processes to ascertain their visual acuity. Not until such work is carried out can anything be known for certain as to the visual needs of different occupations.

Effect of Industry Upon Visual Acuity.

Certain occupations certainly affect sight: at the one extreme there is miners' nystagmus, associated with an extremely low degree of illumination, and at the other glass-blowers' cataract, associated with excessive exposure to rays from molten metal.

What is not clear, however, is the effect, if any, produced by less pronounced conditions. A visitor to a place where close vision is needed for sewing and the like will note that an undue proportion of the workers wear spectacles; and may draw the conclusion that the work has definitely damaged the sight. He may be right; but, first, information is needed

of the visual acuity possessed by the general community at the same age. Here no standard exists referring to a sufficiently large sample distributed by age and sex. Until such a standard comes into existence, the effect of different occupations upon eyesight cannot be evaluated. Such a standard is badly needed; the closest approximation is that for juveniles of school age, but this cannot be fairly applied to adults, especially to those of middle age, which is the class among whom any effect exerted by occupation ought to be most pronounced.

What appears to be clear is that the human eye was not evolved for the purposes for which it is now so constantly used, and that, when it is so used, it is found to be more or less a defective organ. How far this use acts injuriously requires to be determined, so that visual acuity and occupation can be mated.

Summary.

The conclusion of the matter seems to be that while there is a little information, but not much, as to the effect of visual acuity upon industrial efficiency, there is a lack of any sure information as to the effect exerted by industry on visual acuity. In both respects there is a wide field for investigation in order that knowledge may be obtained upon which advice can be given to those possessing different degrees of visual acuity, as to what occupations and processes are suited to their individual capacities.

Opening Paper.

The consequences of myopia as an industrial disease of the eyes.

By N. Bishop Harman.

We have every industry in London, mining alone excepted. But there is no straight run of identical industrial work to give a similar character to our industrial eye diseases except in one condition. A visit to any railway terminus between 9 and 10 a.m. any week day will prove that thousands of our London

citizens are engaged in close work all day. The feature of work in London, as the centre of commerce, is the immense amount of clerical labour involved. Akin to this is the strain on the eyes in the considerable amount of professional and technical work, such as that done by teachers, dentists, compositors, and skilled manual workers at the bench. It is a matter of common observation that continuous close work engenders fatigue, and that the liability is increased where there exist errors of refraction or muscle balance. In some cases there occurs a breakdown in the ability of the worker to continue his work; so that there is a loss to worker, employer and community. Besides this liability to breakdown, there is a lesser degree of crisis, one which makes the worker less efficient in his job; he or she continues at work, but the work done is inefficient as compared with the standard of that same worker under previous conditions of health and of other normal workers. Workers who have experienced these failures desire to obtain a change of work if possible, even at some loss of earning power. But for the most part the sufferer returns to work after an interval of rest, and becomes one of the inefficient workers following a "ca' canny" policy by reason of physical disability.

The economic loss of these cases, the suffering involved, and the unhappiness which the failure to attain engenders in the mind of the willing workers is so great that we should attempt to prevent it. By suitable means those who are likely to fail could be picked out before they make a choice of their life-work, and their energies diverted into more promising channels. In the words of Mr. J. Herbert Fisher, as President of this Society, in his presidential address of 1921: "A little guidance given to the progressive myope about to embark upon the serious business of life, a suggestion as to suitable employment, may save such an one from a life of misery ending in shipwreck."

In giving evidence before the Committee on the Causes and Prevention of Blindness in 1920* I gave an account of

* "The Causes and Prevention of Blindness, based upon an Analysis of 4288 Cases," *Brit. Med. Journ.*, November 5th, 1921, p. 727.

the causes of blindness in 925 private cases, and therein it was found that the consequences of high myopia, over 10 D., accounted for 13·93 per cent., which was the second highest total of blindness, and the highest amongst irremediable causes of blindness. It was further noted that very many of these persons had been engaged in clerical work during their working life.

To determine what, if any, added liability to breakdown or grave defect of vision follows continuous close work by myopes, as compared with the incidence of the same untoward conditions in myopes who were not engaged in such close work, I investigated the records of some 7000 private patients seriatim. These patients were of all classes of life and of varied occupations. From these records there were picked out all those myopes of over 3 D. who were between the ages of 20 and 60 years. Cases of congenital defect, accident or disease unconnected with myopia and of monocular myopia were excluded. The total number of myopes thus collected was 480—quite a sufficient volume of material to allow of reasonable inferences being drawn. The cases were separated into two main sections: (1) habitual close workers, and (2) those not so engaged. In each section there was an age-grouping in decades, and also in groups according to the grade of their myopia—from 3 to 5 D., 5 to 10 D., and over 10 D. In cases of astigmatism the average of the four meridians is taken as the degree of myopia. Note was made of the events of their working history, the occurrence of indications of abnormal stress in work, breakdowns which entailed cessation of work, or change of work where that was possible, or a continuance of the same work under modified conditions; and, lastly, of direct evidence of damage to the eyes. The results thus obtained from the examination of these cases and the comparison of the two sections, the habitual close eye workers, and the other patients who were not close eye workers, are available for comparison (Table I).

Of the 480 cases investigated, 38 per cent. were engaged in habitual close eye work, and 62 per cent. were other patients not occupied in continuous close eye work. The higher

Table I.

Type of patients.	Number.	Breakdowns.	Damage to eyes.	Totals of failures.
Habitual close eye workers	183	70	27	97
Percentage . . .	100	38·2	15	53
Others	297	7	21	28
Percentage . . .	100	2·4	7·06	9·4
Total percentage in 480 (both classes)	100	16	10	26

Table II.

Myopia and type of patient.	Age. 20–30 years.			30–40 years.			40–50 years.			50–60 years.			Totals for type and myopia.		
	Number.	Breakdowns.	Damaged.	Number.	Breakdowns.	Damaged.	Number.	Breakdowns.	Damaged.	Number.	Breakdowns.	Damaged.	Number.	Breakdowns.	Damaged.
−3 to 5 D.:															
Habitual close eye workers	30	8	—	21	9	1	11	2	1	9	2	1	71	21	3
Others . . .	43	—	—	25	—	—	31	—	—	17	—	1	116	—	1
−5 to 10 D.:															
Habitual close eye workers	30	14	2	26	14	2	13	7	2	12	5	3	81	40	9
Others . . .	32	—	1	27	1	1	33	1	1	26	1	1	118	3	4
er 10 D.:															
Habitual close eye workers	5	1	3	9	2	4	9	4	4	8	2	4	31	9	15
Others	11	—	—	15	1	2	16	—	4	21	3	10	63	4	16
Totals for ages .	151	23	6	123	27	10	113	14	12	93	13	20	480	77	48

figure of the second group is accounted for by the fact that it includes a large number of married women, and also most of the very high myopes who were prohibited from doing close eye work. The extent of the failure amongst the habitual close eye workers as compared with the other patients is remarkable. No less than 53 per cent. of the close workers

failed at some time during their career, and of these 15 per cent. sustained permanent damage through the loss of an eye, and two persons lost the sight of both eyes. Amongst the ordinary patients failures were few—only a total of 9·4 per cent.—but amongst these the greater number, 7 per cent., sustained damage to their eyes, and these were very high myopes ; four of these persons lost both eyes.

The second table shows that there is a gradual increase in the incidence of breakdowns and damage to eyes with the increase of age and the grade of myopia. Amongst the ordinary patients there was only one case of trouble at any age amongst those with less than 5 D., but quite a number amongst the habitual close eye workers. The breakdowns and damage increase rapidly for the close eye workers with 5 to 10 D. of myopia, for it is in this group category that they are most numerous ; but the failures amongst the other patients are few only.

Breakdowns and damaged eyes (with one exception of ages 50–60) are actually and relatively most numerous amongst the habitual close eye workers, but it will be noted that there is a higher rate of damaged eyes than of breakdowns amongst the other patients ; this is accounted for by the fact that this section contained most of the very high myopes ; they had been warned off close work, and without doubt attention to this warning had saved many, but some of them had such high degrees of myopia that damage was almost inevitable.

The character of the damage sustained was as follows : Detachment of the retina, 14 cases ; degeneration of the macular region, 34 ; gross vitreous hæmorrhages and the damage consequent thereon, 5 ; rapid myopic cataract, 3. It is noteworthy how much more serious is the risk of loss of central vision from degeneration of the macula in these myopes than is the risk of detachment of the retina. Happily loss of the macula does not leave them in such a hopeless state as does detachment of the retina. Amongst total losses only six cases have been numbered ; but in many others, with the total loss of central vision of one eye there was also

a considerable diminution of the vision of the other eye from the same condition of macular degeneration.

The work done by some of the habitual close eye workers was sometimes of an incredible nature. One subject with 17 D. of myopia was engaged as a printer's proof-reader, and corrected statistical tabular matter all day long; another was a miniature painter with 12 D., and yet another a dental mechanic with 10 D. Some of the close eye workers had made a change of work on their own account; two lawyers forsook their clients, one to become a farmer, the other a cinema controller; the miniature painter had taken over the management of a depot; and the mechanical dentist became a shop assistant. But the proof-reader preferred to continue and take the risk.

The lesson of these figures is plain enough. No matter what view we may hold of the causes of myopia, it is certain that the risks of myopia are heaviest with those patients whose occupation entails habitual close eye work. The risk is so serious that we are in duty bound to fend off young people, who are affected by myopia of anything more than the lesser grades, from work of this kind. The failure to carry on with their work, if it be close eye work, is a serious risk; and the liability to damage to one or both eyes is such that a high percentage of them may sustain partial blindness.

Opening Paper.

Ocular anthrax.

By Charles Killick.

Having had a case of ocular anthrax under my care recently, it occurred to me that it might interest the members of the Congress if I could collect a few details about this, the wool-sorter's disease, a disease which is of necessity unfamiliar to the majority of ophthalmic surgeons. I have myself, during three years' practice in Bradford, only seen the one case, and that only in the later stages, but my friend Dr.

Eurich, who is an authority on anthrax, has had about 250 under his care extending over a period of seventeen years. Of these nineteen were ophthalmic cases, although in six of the latter the site of infection was the eyebrow.

Anthrax in ophthalmic practice is confined to the eyelids or their vicinity. Anthrax of the globe has not, according to Dr. Eurich, hitherto been recorded. As stated above, the eyebrow was the site of infection in six cases, with secondary infiltration, and œdema of the upper lid and extension of the necrosis into it. In one case the pustule was immediately beneath the inner canthus; in the rest it was more or less in the centre of the lid. It is a curious fact that the palpebral border, which is so frequently the seat of infective processes, is rarely, if ever, the starting-point of anthrax.

The appearances of a typical malignant pustule are, of course, well known, namely, a central black slough of variable size, surrounded by a more or less clearly defined ring of vesicles and occupying the centre of an area of a red and brawny inflammatory œdema. The surgeon does not often see the pustule in its earlier stages, when it takes the form of a small papule surmounted later by a small vesicle. Sometimes the papule itself is wanting, a small patch of erythema merely is present, not unlike a flea-bite, surrounding a vesicle so small that close inspection may be necessary to discover it, especially if its contents have escaped. When an eyelid is the seat of anthrax, œdema is more than usually an early manifestation, no doubt owing to the looseness of the tissues involved. This early appearance of œdema is probably the reason why these patients seek medical advice relatively quickly. In over 75 per cent. of the cases of anthrax of the lids a doctor was consulted as early as the second day of disease. In anthrax of the eyebrow advice was not sought quite so soon, and in two cases not until the fourth day. Taking into consideration the very extensive inflammation and œdema (Fig. 1) a fatal termination is much less frequent than one would expect. Of the pustules upon the eyebrows only one caused death, fluid from the vesicles having trickled down the face into the mouth, and having given rise to anthrax

of the stomach. Of the lid cases one died; he was urged by relatives and friends to seek advice, but obstinately refused to do so until the fourth day, poulticing the part meanwhile.

FIG. 1.

Death resulted also in the case of a young man of 27 in whom the pustule was below the inner canthus, but here the real cause of death was not anthrax but salvarsan poisoning, induced, no doubt, by the unsuspected presence of chronic nephritis.

The prognosis, then, in these eye cases is on the whole favourable, owing probably to the early diagnosis, the death-rate of cutaneous anthrax being, according to Dr. Eurich's statistics, 10 per cent. In every instance death occurred as the result of generalised anthrax, and in not a single instance were the orbital tissues manifestly involved.

The diagnosis is a bacteriological one. Reliance must not be placed on the clinical appearances, which are suggestive but not conclusive. A close resemblance to anthrax is often produced by vaccinia. In the case, moreover, of ocular anthrax the patients seek advice so early that the typical anthrax has not had time to develop.

The treatment is comparatively simple. Excision of the infected tissue is, in Dr. Eurich's opinion, inadvisable in the early stages and impracticable in the later. Nor are injections of phenol into the tissues around the pustule of much use, save possibly when the eyebrow is the seat of the trouble. Absolute rest is essential, the patient's head being fixed with the aid of sandbags, while food is given by feeding-cup and spoon. In Bradford anti-anthrax serum is administered in doses of 60 to 80 c.c.: 80 c.c. are given on the first day, and 60 on subsequent occasions, generally at intervals of 24 hours. In some cases as many as 240 c.c. in all have been given. Acting upon a suggestion of Prof. Ehrlich's, Dr. Eurich has given salvarsan in a few of these ophthalmic cases as well as in anthrax originating in other parts, but the number is too small to permit of deductions being drawn from them.

When we consider the frightful appearance presented by some of the patients when the disease is at its height, it is remarkable how small is the cosmetic defect finally left to be dealt with. In many cases some œdema or plastic infiltration alone remains, which, after persisting for months, yet disappears in the end. In the course of a prolonged search through the records of the Royal Eye and Ear Hospital, Bradford, I can only find the notes of two patients upon whom it was subsequently necessary to perform a plastic operation. Both these are to be found in the 1905 records,

and I cannot help thinking that there must have been others; indeed, the Matron tells me that there were, but if so their case-papers must have been lost. Of the two cases, the one was for a protuberant ectropion of the lower lid, but the notes are scanty and the result is not given. In the second case the patient had local anthrax involving the left eyelid. It was treated by serum. The affected portion of the lid formed a dry slough and extensive ectropion resulted, the inner aspect of the upper tarsal cartilage being turned upwards and the conjunctiva of the superior fornix forming a pulpy swelling and so covering the eye. The eyeball and cornea were normal. At the operation the lower lid was first dealt with by excision of adhesions, and two months later the upper lid was restored by a flap taken from above the brow. The result seems to have been satisfactory.

In the one case of my own, œdema of the right eyelids has persisted for months, and was for a long time a considerable disfigurement, especially as the patient was a young woman, but it is slowly passing away. All that now remains of the original lesion is a puckered scar below the eyelid. In this case the optic discs are pale and vision is deficient, but it is doubtful if there is any connection between this and the attack of anthrax.

I will not waste your time by going into the prophylactic measures which have been adopted to minimise the danger of contracting the disease by those engaged in wool-sorting. It suffices to remark that one of these measures was the subject of a recommendation in the Geddes Report, namely, the suggested scrapping of an establishment in Liverpool, where attempts were being made to rid the possibly infected wool from anthrax germs before its distribution to the manufacturers.

My thanks are due to Dr. Eurich for what amounts to almost the whole of the subject-matter of my contribution, and also for the loan of the photographs, etc.

The President

said the Society was greatly indebted to the distinguished

visitors who had brought this aspect of the subject in which the members were interested before them, and a good many questions had already been raised. Many members must be familiar with eye conditions in the industrial districts in which they practised. Mr. Bishop Harman had indicated fairly well the conditions in London and their effect on the eyesight, and the capacity which people with varying degrees of myopia possessed for taking up certain occupations. There was need for more working out in detail, but something had been done in that direction.

Mr. Healy

said he was particularly interested in tin-plate workers' cataract, and there were one or two points in connection with that disease to which he would like to direct attention. One found a posterior cortical type, apart from the posterior polar variety, and one found the posterior polar variety associated with the type Mr. Cridland described, namely, the cortical variety, in the inner and lower cortex. Whatever the cause of the posterior polar variety might be, apparently it was also the cause of the broom-like opacity in the lower and inner cortex. He did not think the direct action on the lens of the heat-rays had much to do with it. He thought, as Hill and Hartridge pointed out in their paper* describing experimental work, to prove the effect of the rays on, and the absorption of heat by, the various media of the eye, that the largest proportion of heat-rays were absorbed by the iris, and he, the speaker, thought the development of the cataract was brought about by the rays absorbed by the iris. From that continual absorption there was caused an alteration in the secretory endothelium of the ciliary processes and in the chemical constitution of the aqueous. It might lead to an increased formation of salts and proteins in the aqueous, thus altering the nourishment of the lens. The absorbed heat might spread along the suspensory ligament to the lens epithelium, causing such damage to the lens epithelium as to admit aqueous by filtration into the lens

* *Proc. Roy. Soc.*, B, vol. lxxxix, 1915.

substance, and thereby swelling and degeneration of the lens fibres, and cataract formation. That was only one suggestion; there were other ideas. We did not know sufficient about the anatomy of the lymph-channels in the eye. It was possible the posterior cortical variety might be produced by some alteration in the contents of an essential lymph-channel which reached the posterior pole. To think that the direct rays could all concentrate on the posterior pole seemed absurd; it seemed to be the visualisation of a mathematical point which had no existence. These rays had to pass through, and would be mostly absorbed by a considerable layer of lens and aqueous matter before reaching the nodal point.

With regard to the general environment of mill-men, he agreed with Mr. Cridland that further investigation should take place as to the effect of manual labour upon the general bodily health. He, the speaker, had one case in mind at the present moment—a roller-man, aged 41. He came to the speaker with left homonymous hemianopia, which came on suddenly. He retained central vision, but the condition never improved. He had had the case under observation six months. In that man no cause could be found for the arterio-sclerosis which he had; his blood-pressure was 205 mm. A Wassermann was done three times, with negative results; and he was examined by several good physicians, who failed to find any cause for the arterio-sclerosis. There was, he thought, a possibility that men who worked in heat and were constantly sweating profusely, and therefore imbibing large quantities of fluid, might through alternative vascular tone develop arterio-sclerosis, and this might be an adjuvant factor in the production of cataract.

Mr. Cridland raised the question of the vision after operation for cataract among iron-workers. He, Dr. Healy, had seen many cases of cataract among iron-workers, and he thought that, on the whole, the results after operation were as good as in the ordinary run of cases of senile cataract; at any rate, he had never been able to discover any gross lesion of the deeper membranes. Recently a case was sent

to him for examination under the Compensation Act, as to whether the condition he had was iron-workers' cataract or not. The man had a typical posterior polar cataract, with, it was true, some striæ in the peripheral cortex ; but he found there was gross degeneration of choroid and retina far forward. He therefore put the case out of court, because he did not think a case of that sort could be one of heat cataract. Any case which showed any other degenerative changes in the back of the eye might be secondary, and could not be put down to heat. He had now examined some hundreds of cases, but had never seen a case where the back of the eye showed gross degenerative changes from exposure to the heat from molten metal.

Another point was one Dr. Legge referred to, namely, that of eye-strain in good visibility and the question of eye-strain in bad visibility, *i. e.* the development of miners' nystagmus because of eye-strain. He, the speaker, did not think nystagmus was due to eye-strain at all, but that it was a physiological effort on the part of the eye to see in conditions of very dim light. The macula ceased to function, and the miner was dependent on his rods, which were organs for the perception of moving objects : so that in order to see in darkness the miner had to move his eyes because the objects looked at were stationary, and the rapid oscillation was an effort on the part of the eye to produce rod vision in the area of the retina where the rods are most congested and the necessary movement least. When the movements continued on the return to normal lighting they became pathological.

Dr. Brinton (Johannesburg)

said that he occasionally met with posterior polar cataract in South Africa among the gold-smelters and the assayers in gold mines. He had one case of posterior polar cataract in a man aged 32, but that disease was not included in their Workmen's Compensation Act. Therefore the speaker had had a chat with the manager of one of the big companies, and he was prepared to accept it when the speaker got a case in which he could say the man's wage-earning capacity

was lessened. So far, all the men he had seen with it had been able to carry on. He was sure an assayer could not carry on his work with an aphakic eye, therefore he must be compensated more than he seemed to be under the English Law, because he was a member of a skilled profession and would have to accept a lesser wage.

With regard to heat cataract, many natives were employed in the work, and they were very accustomed to heat—certainly more than were Europeans, being more pigmented; and he had never come across a case of cataract from heat in a native.

Answering the President, he said the assayer had to examine numerous small samples in crucibles; they took a sample eight or ten feet apart all the way down, sampling the reef, and each one had to be kept in a separate crucible, so that the men were facing the furnace and moving the crucibles for many hours in the day.

He had never met with a case of miners' nystagmus there, though there were 40,000 white men and 150,000 natives in the mines. The gold mines were lighted by electricity.

Dr. Legge

thanked all who, in the discussion, had added to the interest of the afternoon.

Mr. Cridland,

replying to Mr. Healy's comment on his statement that perfect visual results were not obtained after operation, said the results were satisfactory enough, but he meant by a perfect visual result $\frac{6}{6}$; the vision he had obtained was $\frac{6}{9}$ or $\frac{6}{12}$.

He agreed with all that Mr. Healy said; he thought there was a big field for investigation. He could see what was going to happen—that certifying surgeons in industrial districts would be in a very difficult position. Some surgeons would be ready to certify every case of cataract in an iron-worker who might present himself. Then the case might be challenged and sent to the medical referee, and the situation would be difficult. Could members of the Society decide whether it was only the posterior polar form of cataract

which was compensatable? It was not uncommon for those practising in ironworks districts to find a man who was a furnace-worker coming for the first time with a mature cataract in one eye, and with an incipient cataract in the other eye. He thought such a man ought to have compensation, because he considered the condition arose out of his work, but it was not in the category. The profession did not seem to have gone far enough in the investigation to say positively such a man was entitled to compensation. He could conceive a difficult situation in the future in this respect. They all knew that these posterior cortical cataracts could occur in people who had never worked at furnaces. He had the case of an old soldier, aged 70, who was a beautiful example of furnace-workers' cataract. He had lived fifteen years in India. He did not know whether there was any relationship between the two.

The PRESIDENT

thanked Dr. Legge and Prof. Collis for their addresses.

III. DISEASES OF CONJUNCTIVA AND CORNEA.

1. *Corneal ulceration in ophthalmia neonatorum and its after-treatment.*

By M. S. Mayou.

The cornea in ophthalmia neonatorum is affected either by becoming hazy, or by ulceration. The hazy cornea in the early stages is probably only due to œdema of the epithelium, but when the cornea is very hazy, especially when it is localised, the early stages of true infection are present, and corneal ulcers usually follow.

Corneal ulceration.—It is frequently said that if a case of ophthalmia neonatorum is treated sufficiently early and efficiently, corneal ulceration does not occur. This is not strictly true, although it is a very good principle to go on, and applies to gonorrhœal cases in which the baby is healthy and does not get ill during the course of treatment. My own experience is that the general condition of the child is just or almost more important than proper local treatment in the prevention of corneal ulceration. In nearly all cases when corneal ulceration starts, the child has either had some slight illness or is in a debilitated condition. The two principal diseases which lead to debility are gastro-enteritis through improper feeding, and congenital syphilis. In the former instance there is no question that the best way of getting over this difficulty is that the mother should nurse the child herself, and that in the case especially where the mother does not nurse the child, the child should be placed in the best possible hygienic condition both as regards feeding and open-air treatment. For this purpose open-air wards have

been provided at St. Margaret's Hospital with very beneficial results.

Corneal ulcers are of three types :

1. Puncture ulcers.
2. Large ulcers with considerable infiltration of the cornea.
3. Sloughing of the whole of the cornea.

Under these headings the after-treatment is discussed.

1. *Puncture ulcers* are those in which there is a tiny perforation straight through the substance of the cornea, with very little infiltration, and which will develop and run their course to perforation in twenty-four to forty-eight hours, subsequently leading to a very slight nebula, possibly with an anterior polar cataract or adherent iris. The infection is always gonorrhœal.

The children are nearly always strong, healthy infants, and the perforation may be situated in any part of the cornea. The infection in these cases is probably a local one through some small abrasion, and is limited in extent because the nutrition of the cornea is good and the resisting power high.

I have never seen a case of anterior synechia from an ulcer of this type give rise to any trouble. It is probably quite unnecessary to perform any operation with a view to detaching the iris from the back of the cornea; on the other hand, an anterior polar cataract may sometimes require operative treatment, although, as a rule, they are best left alone. Optical iridectomy, as far as my experience goes, has not been very satisfactory in improving the vision. On the other hand, needling in some cases has yielded a satisfactory result. In a child of twelve, with $\frac{6}{24}$ vision in both eyes and $\frac{6}{18}$ with a dilated pupil and an anterior polar cataract practically filling the pupil, at the request of the parents the one lens was needled, and she subsequently got $\frac{6}{9}$ vision, which enabled her to do far better in her school than she had been doing previously.

In the next two groups of cases, corneal ulceration occurs in rather more than 75 per cent.: it begins at the junction of the lower third with the upper two-thirds of the cornea.

Why the ulcers should so frequently start in this part of the cornea is a little difficult to make out. Although in adults it is the most exposed part of the cornea and therefore liable to injury, in the infant with ophthalmia neonatorum the eyelids are closed, but it is the portion of the cornea which falls opposite the line of union of the lid, and it may be due to the tightness of the lid margin pressing against the cornea giving rise to some slight damage of the epithelium which causes the ulcer to start at this situation.

2. *A largish ulcer with considerable infiltration of the cornea.*—In the early stages these may present themselves as accumulations of pus beneath the epithelium or a breaking-down hazy cornea. They frequently perforate, leading to dense nebulæ with an adherent iris.

It is these cases that one usually has to do something for after the ulcer has healed. After they leave the hospital they are given a lotion, and yellow oxide of mercury ointment to use, and operative treatment is avoided—at any rate until the child is weaned—as the corneal scars will often clear up in an extraordinary way. The exception to this is where there are dense corneal scars in both eyes with obviously very little vision. The indication of this is nystagmus. Nystagmus in these children starts directly the eyelids begin to open. There is no nystagmus before, even if both corneæ are destroyed by corneal ulceration. In a case where one eye was destroyed by a corneal ulcer and the other one remained clear, nystagmus persisted on the side of the blind eye for a year and a half, but there was no nystagmus on the other side, even by examination with the ophthalmoscope. Cases where nystagmus is present should be operated on as early as is consistent with the general condition of the child. Where the corneal opacity occupies the central area of the cornea it is obvious that some form of optical iridectomy must be performed, and that the clearest part of the cornea must be selected for the purpose. According to the text-books, where it is possible to make a choice of the situation, down and in is usually selected. My experience is that this is not at all a good situation. The great trouble with a

patient after an optical iridectomy has been performed is that he suffers from too much light being admitted to the eye. If an optical iridectomy is done downwards and inwards there is no means of cutting off the excessive size of the aperture by means of the eyelids, whereas if the iridectomy is done either directly inwards or directly outwards, the upper lid can be closed over the aperture so as to decrease its size. The smaller the iridectomy the better the result obtained therefrom if the cornea be clear. Personally I use a keratome, making the incision inwards, seize the iris with a pair of forceps about 2 mm. from the pupillary margin, pull it out and snip a small piece out of it, holding the iris scissors at right angles to the incision, and try to leave the sphincter of the iris intact.

Where the corneal opacity occupies very nearly the whole cornea, and there is only a clear rim of corneal tissue around the margin of the cornea, if one tries to obtain an iridectomy in the manner above described one finds first of all that the iris will as a rule be so adherent to the scar that when it is pulled upon it tears, and an imperfect and unsatisfactory iridectomy is the result, or the incision which is made at the limbus leads to an opacity in the cornea, with the result that if a more or less satisfactory iridectomy be obtained, its result is made negative by the opacity from the incision. In these cases also the iris is completely drawn forward, and they are liable to develop secondary buphthalmos from tension. To remedy this I perform iridectomy from the centre of scar. The keratome is inserted from the middle of the cornea and made to appear in the anterior chamber, the iris forceps are introduced and the iris seized in its periphery and torn away at the angle of the anterior chamber. There is practically no danger of wounding the lens, as although the iris is drawn forward, the lens is in its usual position, although one cannot see the point of the knife when first it is inserted. A good opening in the iris is usually the result, if the iris is not too atrophic.

The results of optical iridectomy vary. If there is a sound eye on one side an optical iridectomy on the other probably

will only improve the vision of that eye for large objects; on the other hand, if the one eye is lost, an optical iridectomy on the other will often improve the vision markedly. In one such case where an optical iridectomy outwards was performed the child could fix objects, but to do so he turned the eye inwards so that it looked as if he brought the aperture to bear on the object looked at. Sir John Parsons' explanation of this is that the eye was very hypermetropic owing to flattening of the cornea and the lens, and to get the peripheral rays to fall on the macula the eye had to be turned inwards.

Tattooing of the cornea is most useful in making the appearance of the eye much better in cases where there is a very large obvious white scar. It has also been used in the case of an absolutely small dense white opacity in the centre of the cornea after doing an optical iridectomy inwards. In one such case the patient was a boy of about sixteen, and he said it improved the sharpness of his vision considerably.

Cases of tattooing have been followed by sympathetic ophthalmia, so that the operation should be reserved for those cases in which the scar is practically free of iris tissue.

3. *Sloughing of the whole of the cornea.*—This type of ulcer occurs in children who are marasmic, either as the result of gastro-enteritis, congenital syphilis, and especially in premature children. It generally starts with a dense infiltration of the whole of the cornea, the central portion becoming necrotic and breaking down. In some cases there is a fair amount of discharge, and in other cases there is extremely little. The condition is practically one of keratomalacia.

Sloughing of the cornea is frequently followed by extrusion of the lens in its capsule through the opening, and the vitreous may subsequently present.

In gonorrhœal cases I have never seen panophthalmitis follow perforation either with or without extrusion of the lens; in streptococcal and staphylococcal *aureus* infection panophthalmitis does occur.

At St. Margaret's, after the lens has been extruded, nothing is done beyond the ordinary routine treatment and the ulcer is allowed to heal.

Where panophthalmitis is suspected or has occurred, evisceration of the globe is performed through the ulcer after enlarging the aperture.

Extrusion of the lens is very important, especially as regards the prognosis of the case. The umbo of the lens presents in the base of the ulcer as the result of crying or straining, the lens is delivered through the ulcer, and generally remains attached by a few fibres of the suspensory ligament, which are then torn through by the closure of the eyelids.

In cases in which the lens has been extruded it is always followed by secondary buphthalmos. This is due to the vitreous and iris being incorporated in the wound causing complete obliteration of the anterior chamber. As yet no means has been discovered of preventing it, and it usually means that the eye has to be eviscerated at a later date and a glass ball inserted, taking care to stitch both the sclerotic and the conjunctiva separately with silk sutures.

It is extraordinary how rapidly the corneal ulcers will heal even when the whole cornea is involved. This is probably due to there being a freer circulation of lymph between the layers of the cornea in the child than in the adult: as a result, the new cicatricial cornea is quite as thick and sometimes thicker than the normal cornea, both in the new substantia propria and epithelium. Large granulomatous masses are not infrequently present in the course of healing.

The PRESIDENT said that the variety of optical iridectomy suggested as appropriate to anterior synechia, with a limited peripheral portion of clear cornea, was a new and promising idea which many members might have an opportunity of trying.

Mr. HARRISON BUTLER said that in the East, where he had practised for some years, they saw large numbers of perforations practically all from non-venereal ulcers. He never saw any septic trouble associated with the perforation, and there was no case of pan-ophthalmitis. There was one of sympathetic cyclitis, but in this case a Guthrie's section had been

made. Anterior synechiæ were very common, but they seemed to cause remarkably little irritation. He thought that too much stress had been laid upon the irritation caused by the drag of an anterior synechia. In this respect they differed from posterior synechiæ, which did sometimes appear to favour recurrent iritis. He thought more trouble was likely to follow operations for relief of an anterior synechia than that caused by the adhesion. These operations were very difficult and often the result was poor. In the East he performed a large number of operations for artificial pupil: in fact, after trichiasis, it was the commonest operation at the British Ophthalmic Hospital. The so-called site of election down and in was generally impossible, because the nebula was generally most developed in this area. He generally chose the situation recommended by Mr. Mayou, outwards. The optical result was generally most disappointing. The most that could be expected was that the patient might be able to get about. It was not in accordance with the laws of optics that the eye must be tilted to get good vision. If a diaphragm be placed in front of a lens, the pencil of light entering by a hole in any part would reach the focus.

Lt.-Col. A. E. J. Lister said cases of ophthalmia neonatorum and similar conditions were very common in the East, where he had practised. He supported Mr. Mayou in his suggestion about the site for optical iridectomy. He had done many optical iridectomies at the site of election, and some people had complained very much of the glare afterwards. As some members might be going to the East, it might be a point which it was important for them to know. If one did an iridectomy the lower edge of which reached the horizontal line approximately, the coloboma could be covered at will by the upper lid, and the size of the pupil varied, which was a great advantage. With regard to the effects of the anterior synechia after a perforating ulcer, he agreed that, on the whole, he had not seen many bad results, but he had seen many cases of secondary glaucoma. He had seen such cases in adults after a perforating ulcer, and preferred

to keep such cases under observation. With an out-patient attendance of about 150 a day a good many cases were seen in a year.

Mr. Johnson Taylor desired to mention a method of making an artificial pupil, used by Sir Anderson Critchett, viz. by means of blunt hook instead of iris forceps. He, the speaker, thought the difficulty in doing optical iridectomy was to remove a sufficiently small piece of iris, and as near the pupillary margin as possible, provided one cleared the corneal opacity; the hook could not be used where there were a large number of posterior synechiæ, but only where there were none, or not many. It was better to do optical iridectomy with Bell Taylor's small angular keratome, which was introduced through the cornea. One could then get a smaller and more circular and a neater iridectomy than with forceps.

Sir John Parsons said he felt diffident in speaking on optics before one of the greatest authorities in Europe. Members were familiar with the point Mr. Harrison Butler had drawn attention to, but owing to the pathological condition the cornea and lens were flattened and the approximate focus was deviated from the ordinary position; hence the deviation of the eye.

2. *An incident in the life history of the xerosis bacillus.*

By M. S. Mayou.

In 1903 I brought before this Society a paper on "Xerosis of the Conjunctiva," pointing out that the condition was due to keratinisation of the epithelium as the result of dehydration, principally from exposure, and that, as the result of the altered surface tension, the tears would not adhere to it, but the bubbles of the fatty Meibomian secretion did so. In this secretion the so-called xerosis bacillus could be found. It also pointed out that the bacillus could not be the cause of the disease firstly, because it cannot be found in the

tissues; secondly, that it can be found in normal Meibomian secretion and beaten-up secretion found at the angles in the lid in chronic conjunctivitis, and it has also been isolated in a large number of cases of chalazion; thirdly, its behaviour in culture is that of a saprophyte; and fourthly, when inoculated in a man or animal, it does not reproduce the disease. It can therefore be looked upon as a saprophytic organism occurring in the Meibomian secretion, comparable to such organisms as the smegma bacillus occurring in the secretion of the glands of the prepuce. It is a little difficult at first sight to say why the organism can be found in nearly all Meibomian secretion, but I think that the infection of the Meibomian secretion with the organism takes place very frequently at the time of birth. In the examination of the discharge from cases of ophthalmia neonatorum one comes across this bacillus extremely frequently. It is found in little masses surrounded by a badly staining area due to the greasy secretion in which it is found. In cultivations from the cases of ophthalmia neonatorum one finds it in as high a number as 70 per cent. of the cases, although in smear preparations one does not find it so frequently. In the vagina there is a pseudo-diphtheritic bacillus which exhibits almost exactly the same characteristics as the xerosis bacillus, except one character on cultivation referred to later. On examination of the vaginal discharge of the mother and the eye discharge of the child in six cases, we have been able to find the bacillus exhibiting identical characteristics in each instance, and there is practically no doubt that the child's eyes were infected from the vagina of the mother. I have found the bacillus as early as the third day after birth, and there is very little doubt that in all the cases the infection took place at the same time as that causing the ophthalmia neonatorum. In text-books it is said that the xerosis bacillus as obtained from the conjunctiva is supposed to be differentiated from the Hoffmann bacillus (another bacillus belonging to the diphtheroid group) and the bacillus found in the vagina, by its growth on agar. It is said that the xerosis bacillus grows in small round colonies, whereas the Hoffmann bacillus

grows as a flocculent mass on the surface of the agar. This is not entirely true, because one can find the xerosis bacillus in these children growing in both manners, and this is probably due to the fact that it is only after it has been growing in the conjunctiva for some time that it takes on the small discrete growths which are supposed to be characteristic of it. As a rule the bacillus as found in the vagina grows in a flocculent form, but it can also be found on culture in discrete colonies. As this is the only point of difference, it may be regarded as an identical organism.

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3. *The analysis of a series of cases of interstitial keratitis.*

By J. F. Cunningham.

This series of cases was started at Mr. Nettleship's request. The object was to collect a large number of cases of interstitial keratitis, with a view to ascertaining the incidence of the disease in the two sexes, the age of onset, and the interval between the onset of the disease in the two eyes. I also kept a column in my records for other evidences of congenital syphilis, and for the family history when obtainable.

It was originally hoped to get 1000 cases, but this was reduced by Mr. Nettleship to an attempt to get 500 cases some time in 1914.

On Mr. Nettleship's death the records were returned to me by his trustees, with the request that I should use them. By the end of 1914 other things had happened, which may in part account for the delay in bringing forward the cases. The cases are drawn from a wide source. They were under the care of Mr. Nettleship, Mr. Lawford, and Mr. Fisher, at St. Thomas's and at the Royal London Ophthalmic Hospitals, and under Mr. William Lang at Moorfields.

There were some of Sir William Lister's cases from the London Hospital, and my own cases mainly from the Central London Ophthalmic Hospital, together with some of Mr.

Juler's cases. Mr. Lawford has kindly allowed me to use data from his private notes.

Dr. W. W. Sinclair is good enough to let me use the figures he collected for Mr. Nettleship as to the incidence of the disease in the two sexes, and I am also using those collected by the late Mr. Ilbert Hancock relating to this point.

I wish to tender my best thanks to those who have helped in this way.

I have collected 435 cases myself, 34 of which I have ruled out as doubtful, leaving a total of 401 cases.

Of the doubtful cases it may be mentioned that nine were probably secondary to acquired syphilis, two were tubercular, and in one case it was uncertain whether the cause was sympathetic ophthalmia or congenital syphilis.

In seven of the 401 cases the attack appears to have been precipitated by injury. I am afraid I have not searched closely for the possibility of the attack being started by an acute illness.

Dr. Sinclair's cases number 111, collected from 10,054 out-patients, *i. e.* over 1 per cent. of this total.

Mr. Hancock's cases were collected with regard to the question of sex alone.

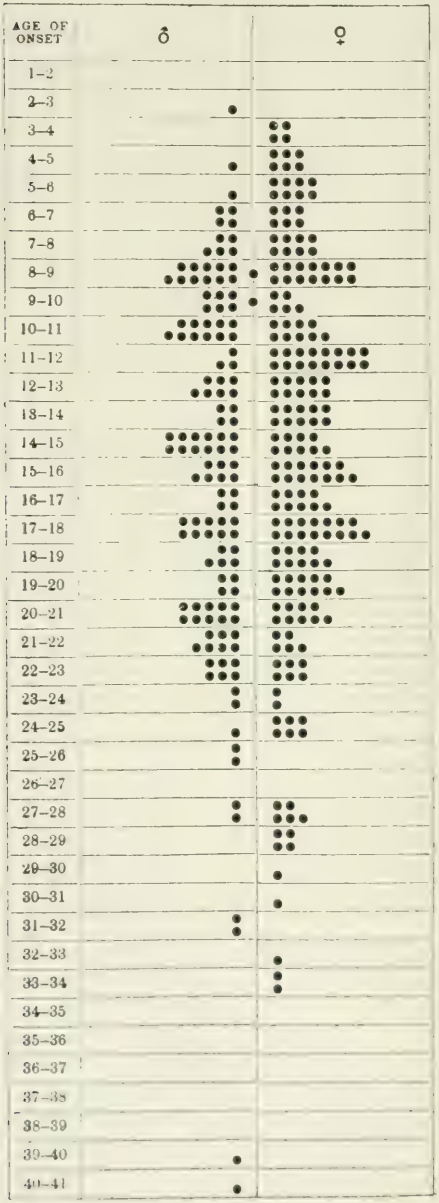
The cases may be tabulated as follows as regards sex :

	♂	♀	Total.
Present series . . .	155	242	397
Dr. Sinclair's cases . .	52	59	111
Mr. Hancock's cases . .	32	41	73
	239	342	581

In the cases I have collected the females exceed the males by ten more than half as many again, whereas in the combined totals they fall short of this by sixteen cases.

Hutchinson, in *Syphilitic Diseases of the Ear and Eye*, found the ratio of males to females in 102 cases as 1 is to 1·7.

In 336 cases I have a record of the age of onset of the disease in the first eye, which is shown in the accompanying diagram (p. 46).


AGE OF ONSET
♂
♀
1–2
2–3
3–4
4–5
5–6
6–7
7–8
8–9
9–10
10–11
11–12
12–13
13–14
14–15
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33–34
34–35
35–36
36–37
37–38
38–39
39–40
40–41

In two cases the sex could not be determined because the Christian name was not entered in full.

The youngest case recorded is a male, aged 2 years and 8 months, from Mr. Lawford's private case-book. Both corneæ were affected, and the forehead was bossy. The first child, born seven years previously, had died in convulsions, aged 7 months. Hutchinson has recorded a case 1 year old at time of onset (Case 19 in his table).

The oldest case in the series is a male, aged 40 years, seen by Mr. Nettleship at St. Thomas's Hospital in 1888. He had double interstitial keratitis of five months' duration. His physiognomy and teeth were typical of congenital syphilis, and he had some scarring about the mouth. The notes are in Mr. Nettleship's own handwriting.

The male, æt. 39 years, was an in-patient at St. Thomas's Hospital under Mr. Nettleship, and also had Hutchinsonian teeth and disseminated choroiditis.

Stevenson quotes Marcus Gunn as recording a case 36 years old at the time of onset, and mentioning two others, 37½ and 38 years old respectively at the time of onset.

Age of Onset.

As regards the age of onset, my figures tend to put the majority of cases as occurring later than in Hutchinson's or in Stephenson's series.

The greatest number of cases are between 10 and 15 years, and this is in agreement with Hutchinson's findings in his 102 cases. Stephenson, in his 97 cases, found the largest numbers between 5 and 10 years. Both of these observers, however, find a higher percentage of cases between 5 and 10 years than is shown in the table on p. 48.

Interval.

In two cases it is recorded that "both eyes came bad together."

There are 15 cases with an interval of 3 weeks and 14 with an interval of 2 weeks, *i. e.* the greatest numbers of cases have intervals of 2 or 3 weeks. Thirty-eight cases, *i. e.*

exactly one-third of the total, are recorded in which the interval between the two eyes ranged from 2 to 5 weeks, and in more than half the cases the interval was under 11 weeks.

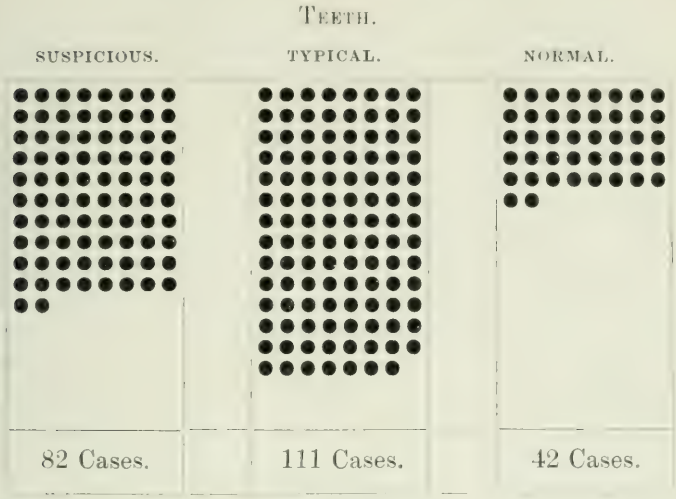
Age of onset (years).	No. of cases.	Percentages.
1–5	12	3·5
5–10	70	20·8
10–15	91	27·2
15–20	87	26·0
20–25	54	16·1
25–30	14	4·1
30 onwards	8	2·3
Total	336	

There are 14 cases in which the second eye was not attacked for more than a year after the first, the intervals being—one at 1 year and 1 month, two at 1 year and 2 months, two at 1 year and 6 months, one at 2 years, one at 3 years, one at 3 years and 9 months, one at 4 years, one at 4 years and 2 months, one at 4 years and 7 months, one at 5 years and 11½ months, one at 6 years, and one at 19 years.

The longest interval I have found is 19 years. This was in a case under Mr. Juler at the Central London Ophthalmic Hospital in 1914. The right eye was affected when she was 12 years old, and the left was not attacked till 1913, when she was 31 years of age. We had, however, to rely on the patient's statement as regards dates, as we had not notes of the first attack. The teeth are noted as "suspicious," and there were choroidal changes.

Other Evidences of Congenital Syphilis.

Teeth.—I have notes of 235 cases in which the condition of the teeth was recorded. They were described as being typically Hutchinsonian in 111 cases, "suspicious" or "suggestive" in 82 cases, and normal in 42 cases, *i. e.* the teeth led to the suspicion of congenital syphilis in just over 82 per cent. of the cases.



Choroiditis.—In 82 cases there are notes as to the presence or absence of choroidal changes. This, though useful as corroborative evidence, is at best almost valueless for statistical purposes. In 56 cases choroidal changes were noted. In 26 cases no choroidal changes could be seen, although the note frequently applied to only one eye, as it was impossible to examine the fundus in the other, *i. e.* over 68 per cent. of the cases in which an examination was possible showed choroidal changes.

Deafness was noted in 36 cases.

Physiognomy.—In 79 cases this was noted, and in 65 of these cases it was typical or suggestive of congenital syphilis, in 14 cases it was recorded as "not," or "not at all suggestive" of this disease, *i. e.* in the cases recorded the facies was typical or suggestive in 82·27 per cent.

There are 11 cases in which nodes are recorded, 12 in which there was synovitis of the knee-joints, 10 in which the palate and 1 in which the larynx was affected. I think, however, it is pretty certain that the palate and fauces are not looked at as frequently as the teeth. Or, to put it another way,

the evidence necessary having been found in the central incisors, the mouth is not explored further.

Family History.

There are 83 cases in which there is a record of the family history.

I have notes of three instances in which two members of the same family were affected, and nine other instances in which more than one member of the same family were said, on fairly reliable evidence, to have the same disease. In one case the statement is made that the patient "has four brothers and four sisters, and that they all have bad eyes."

The number, 12 families, in which more than one member is affected, is probably an understatement and valueless, for in very many cases the family history is not recorded.

Of the 83 cases in which the family history is recorded 68 have a history of one or more miscarriages, or deaths in infancy, and 15 have a fairly reliable "good" record as regards the childship of which the patient is a member, *i. e.* 81·92 per cent. have a bad family history, and 18·07 per cent. have a good family history. It is impossible to get the history entire unless the mother is actually seen, and even then the lapse of time has often blurred her memory —at least in the hospital class of patient.

Many of these histories are striking. I give some instances:

Case 10.—"Mother has had six pregnancies. Patient is the eldest. None of the others lived more than $1\frac{1}{2}$ years. One was stillborn. There has been one miscarriage."

Case 11.—"Four living out of a childship of 13. Patient is the youngest of these. Nine died in infancy. One or two miscarriages besides."

Case 17.—"Second in a childship of 10. Others all dead."

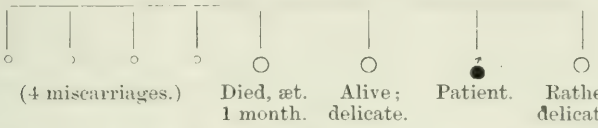
Case 93.—"Mother married three times. First family, 5 in childship; 3 died young. Second family, 5 in childship; 3 died young. Third family, 7 in childship; 4 died young. Patient is the eldest of the second family. There were also three miscarriages."

Cases 135 and 136.—Sisters—under Sir William Lister. Five living.

Six dead or born dead and three miscarriages, besides the living sibs in the tree.

Between the two affected girls, 1 born dead, 1 lived 9 months, 1 miscarriage.

Case 292.—An out-patient under Mr. Nettleship, æt. 14 years.



Case 375.—Male, æt. 14 years, bridge of nose flattened, teeth suggestive, developed synovitis of right knee while in hospital.

One of a family of 14, of whom 10 are dead, one brother blind, one a cripple.

The two other series of cases which I have quoted are Sir Jonathan Hutchinson's series of 102 cases in *Syphilitic Diseases of the Eye and Ear* (1863), and Mr. Sydney Stephenson's series of 101 cases published in his paper on "Interstitial Keratitis from a Modern Standpoint," 1908.

These two observers collected their own cases. I am, of course, largely indebted to the records of others whose notes I have collated.

I feel only too conscious how much more could have been made of this material if it had been arranged by the master mind. I feel it a great privilege that I have been allowed to carry on, however imperfectly, a piece of work which was initiated by Mr. Nettleship.

The President said the Society was very greatly indebted to Mr. Cunningham for this interesting and excellent paper, and especially for carrying on the Nettleship tradition in this

laborious piece of work, and for reducing to figures the facts about so large a number of cases of interstitial keratitis. He, the President, was interested to see that among the latest ages in the large number of cases of inherited syphilis who developed interstitial keratitis which Mr. Cunningham showed was a man in his 41st year. He, the speaker, had himself knowledge of a case aged nearer 50 than 40, in a congenital syphilitic, when the interstitial keratitis came on in the first eye. And in that case the evidences of congenital syphilis were sufficiently well marked. There was some gummatous perforation of the palate. He remembered that case not only because of the late age at which the disease took place, but he learned that the man's father was supposed to be dying of cancer of the rectum, and that might have been the disease, but as the son was a congenital syphilitic, the speaker wrote to his father's medical man and informed him the son was the subject of congenital syphilis, and that the father's rectal disease might have been syphilitic, though it might have later passed into a cancerous condition.

The interval between the incidence of interstitial keratitis in the two eyes had always seemed to him to be very much more extended in adult cases ; it was often an interval, not of months, but of years, before the implication of the second eye.

The present statistics on interstitial keratitis ought to be extremely valuable, and the point which had not been brought out in them—an extremely important one—was as to whether or not interstitial keratitis was initiated by injury. That question came out in regard to compensation, especially in the case of adult workers. They were so prone to allege injury that the mere fact that they had received an injury seemed to be insufficient in a congenital syphilitic with interstitial keratitis to incur the employer in liability on that account. The onset was insidious ; the eye became uncomfortable, there was some redness, and the man thought something had entered the eye. He did not come for advice until some days later, when the symptoms were more intense. Unless some competent person had seen an abrasion, or had removed a foreign body from the eye, the statement that he

had got something into the eye could not be accepted. He only drew attention to that fact because he had heard of an ophthalmic surgeon who was prepared to accept a statement of that kind, and to report that the man would not have developed this unfortunate disaster—which was a very important one from an economic point of view—had he not received this accident. Seeing that the other eye was liable to be affected, it became a more important matter, and if that position were accepted, it would be a very important matter from the trade point of view.

Mr. Cyril Walker said he was very much interested in interstitial keratitis. When he started practice in Bristol, he was struck, almost at once, by the great number of cases, their severity, and the comparatively erratic course many of them ran; moreover, the age at which they appeared was not typical. What the President had said about injury as a determinant was another feature which had interested him, as it had others also, very much. Some years ago he met, at the Eye Hospital, Bristol, two cases. The first was that of a man whose eye had been whipped, rather than scratched, with a piece of wire. There was a very slight abrasion of the cornea, with much redness, some evidence of bruising, and ciliary injection. This stage passed, and he came again in a fortnight with a condition which was suspicious of interstitial keratitis, and a week later still it was definitely that condition.

Another case was that of a man who had been pulling out a nail with pincers, and the nail broke and the pincers bumped his eye, causing a contusion. He attended the hospital with a bruised eye and some abrasion of the lid; he had forgotten whether the cornea was involved. In three weeks he also had evidence of early interstitial keratitis in that eye.

He also remembered a third case, in a woman æt. 27, who had had interstitial keratitis in one eye some time previously. She came on account of a disfiguring leucoma. Having at that time more leisure and enthusiasm than discretion, he was led to tattoo it. The result was good cosmetically, but a year afterwards he saw her with interstitial keratitis in the other eye. On inquiring more minutely he found

that the first attack of interstitial keratitis in the right eye coincided with a pregnancy, and the attack in the other eye corresponded with the date of a pregnancy three years afterwards. [The PRESIDENT: The second eye goes through an attack without apparently receiving an injury.] Mr. Walker replied that this had always been a problem to him. He had also been surprised that other things besides injury did not cause interstitial keratitis—for instance, an attack of conjunctivitis. Three years ago he had a case, sent him by a doctor, of very severe muco-purulent conjunctivitis. He did not know what organism was found. The severe conjunctivitis was almost recovered from, when an absolutely typical interstitial keratitis followed, and the teeth were typical of syphilis, though he did not know whether there was other evidence of congenital syphilis. The Wassermann was negative.

Mr. N. BISHOP HARMAN said the age of onset of interstitial keratitis was very important, as also was the influence it exerted on the returns received from various blind institutions. He recently saw a return from a dozen blind schools, and ophthalmia neonatorum in one institution was given as high as 50 per cent.; and yet another institution gave a return of only 10 per cent. for that disease, and there were all intermediaries. The reason for this great discrepancy was the age at which the children were taken into these institutions. An institution which was established to take in babies got ophthalmia neonatorum as the main condition, so also did an institution which was voluntarily supported and was run by sympathetic people who naturally enough were most interested in the very young, amongst whom the most were blind from ophthalmia neonatorum. Educational institutions which had to take blind children of all ages had a much larger proportion of blindness due to syphilitic disease. These cases did not begin until about 10 years of age, reaching their maximum at 15. In these schools syphilitic blindness reached the high total of 35 per cent. That was an explanation of the big discrepancy in statistics which should be widely known.

With regard to the typical characteristics, he did not think Mr. Cunningham could have seen a paper which he, the speaker, published, giving a record of blind children, in which he gave the condition of the teeth, physiognomy, and scarring of the mouth; it was amazing what a large proportion showed these characteristic signs. The ophthalmic surgeon appeared to see them more than did the general surgeon. He took a well-known general surgeon to one of his, the speaker's, schools, and put the children in a row for inspection, and that visitor confessed he had never seen such an exhibition of syphilitic characters in his life before.

With regard to family history, he collected the life-histories of 150 of these syphilitic families, and put beside them the histories of 150 ordinary hospital mothers. The 150 syphilitic mothers had had 1001 pregnancies, the healthy mothers only 826. But out of the 1001 the syphilitics got only 390 presumably healthy children, whilst the 826 non-syphilitics produced 654 healthy children, showing the great loss in vitality caused by the syphilitic infection. (*Vide Final Report Royal Commission on Venereal Diseases*, 1916, p. 149 *et seq.*)

Mr. Johnson Taylor said the starting of interstitial keratitis was a very practical point. He remembered a case, about 30 years ago, of a painter who, when scraping hard paint off some boards, received a slight injury from a piece of hard material. Before long he had an attack of interstitial keratitis. He came to see the speaker, and was much annoyed when told he, Mr. Taylor, could not sign the certificate stating that the eye was brought into that state by the injury. Still it was a practical question: Why was injury followed by interstitial keratitis? Was it a parallel case to gout? A person with gout in his system would strain his leg or get a kick on it, and he would then get an outbreak of gout in the injured site. Did the injury bring that out? Would it have happened had there been no injury? The interstitial keratitis in that case followed the usual course, and in a few weeks the other eye was involved with the same condition. Had the injury anything to do with that?

Another point Mr. Cunningham brought out was the extraordinary preponderance of interstitial keratitis in female children. He believed it was commonly stated that 103 males were born to every 100 females. Whether it was that the males died off rapidly after five or six years so that there was a marked preponderance of female children at that age—5 to 15—when interstitial keratitis came on, or whether it was that the male children were supposed to be stronger and there was in them greater resisting power, he could not say, but he would like to hear Mr. Cunningham's explanation of it.

Mr. W. T. Holmes Spicer said he had records of 700 cases of interstitial keratitis, and some interesting facts came out from an analysis of these cases, especially in regard to injury. He found that 3 per cent. of cases of interstitial keratitis were directly associated with injury, *i. e.* cases in which there was an intimate relationship between injury and the attack of interstitial keratitis. The kind of thing which occurred was the following.: A man got a foreign body in his cornea; it was removed, but the eye remained irritable, and, in spite of treatment, the case passed gradually into one of interstitial keratitis. There was direct continuity between the injury and this sequel. There were some facts which, he thought, threw light on this: it must be assumed that the attack of interstitial keratitis was due to the presence of the spirochæte somewhere in the body; probably it was in the ciliary body or the cornea, and it lay there latent, and required a certain degree of maturity before it could develop and manifest itself; if an injury occurred to the eye of a syphilitic person, it would not necessarily produce interstitial keratitis, *i. e.* it would not do so unless the organism in that person were present and ready to burst out into activity.

He had had two rather curious results. One was a needling of a congenital cataract: that was followed at once by a very severe interstitial keratitis. No one at first knew what was the reason of the inflammatory attack: but as the case went on and developed, it became clear that it was interstitial keratitis, probably directly promoted by the injury.

At Moorfields he saw a similar case, a needling followed by interstitial keratitis, under the care of Mr. Fleming.

In yet another case he, the speaker, needled a lens; the operation was completed without any attack of interstitial keratitis and the sight became good. But three years later that person had interstitial keratitis.

In one of these sets of cases the injury was directly followed by interstitial keratitis; in the other there was an interval between the injury and the onset of the disease. In the first set the organism had reached maturity and was ready to break out, and in the other it had not reached maturity at the time of the injury. This also explained the difficulty as to the condition coming on in both eyes. It was easy to understand interstitial keratitis occurring in an injured eye, but how could its occurrence in the other eye be explained? Interstitial keratitis was a binocular disease, but the attacks need not be simultaneous in the two eyes. If both were attacked together, the conditions favouring growth were present in both eyes. If there was an interval, then the condition of maturity was not reached in the two eyes together.

The PRESIDENT said the hypothesis advanced by Mr. Holmes Spicer was a very interesting one, and in such a prolific disease from congenital syphilis as this it would be surprising if, in 700 cases in his own experience, Mr. Spicer had not been sometimes caught out in operating for congenital cataract when the spirochæte was ready to burst into activity. Mr. Spicer presupposed that in the second eye the spirochæte was not ready, and that therefore there was an interval. But that did not satisfy him, the speaker, that the injury produced the interstitial keratitis. Even if the needling had not been done, the spirochæte being ready it would have exploded. [Mr. SPICER: I agree.] He did not think this Society, as a scientific body, should allow it to go forward that injury was the exciting cause, and that liability in cases of interstitial keratitis should be incurred in consequence of that. Facts were valuable, but a hypothesis on the matter might be dangerous.

Mr. STACK said he had a patient, a boy, æt. 12 years, who

had just recovered from a bad attack, and his second eye had now got well. The beginning of the attack was interesting. He operated on one eye for squint, advancing the external rectus. Three weeks later he came again, and his squint was doing fairly well, but was not cured. He was thinking of operating on the other eye, but decided to postpone it a week or two. The next week the boy was brought and was starting a very definite interstitial keratitis, and perhaps luckily for the speaker it began in the eye on which he had not operated. In both the boy and his mother the Wassermann was positive; the father was not tested. He also asked whether Mr. Cunningham had had a case of interstitial keratitis in one eye only.

Mr. Healy said he would mention two cases which were of interest in view of Mr. Holmes Spicer's remarks about the spirochæte being latent in corneal tissue. One of the cases he had at present under treatment. The patient, a boy, æt. 12, developed interstitial keratitis in the left eye. The Wassermann was positive, and his mother and father also had a positive Wassermann. The boy was placed under treatment by neosalvarsan and mercury, and at the end of the first course of treatment the Wassermann was still positive. After a three-weeks' rest he was put on to a second course, and eleven weeks after the onset of the keratitis in the first eye the disease started in the fellow eye, whilst he was undergoing the second course of treatment.

The other case was in a man, æt. 42 years, and with him the same thing happened. In his case the second eye was involved seven months after the first.

Mr. Harrison Butler said that he was interested in the relation between accident and interstitial keratitis.

The matter had been brought forward some years ago at the Oxford Congress by Mr. Coulter, who gave details of two compensation cases in which interstitial keratitis followed an accident. Then he had a case in which he had extracted a cataract from a woman who had years before suffered from interstitial keratitis. The operation was followed by a recurrence of the keratitis, which began, not at the site of

operation, but at the centre of the cornea. This eye had to be excised. It was certain that the trauma of the operation had caused the outbreak of keratitis. Shortly after he was asked to report upon the case of a girl who came to the Birmingham Eye Hospital with a small foreign body in the cornea. Interstitial keratitis appeared in the injured eye, and the other rapidly followed. He reported that although the disease was due ultimately to congenital trouble, yet the injury was the deciding factor. He had since then had several compensation cases of a similar type, and he reported that the employer is liable for the keratitis.

Last week he had seen a child at his School Clinic who had a normal cornea. Atropine was instilled in the usual manner. A week later he saw the child with typical interstitial keratitis. He would not labour this case, which may have been an example of coincidence, but he thought himself that the slight trauma of the instillation had caused the outbreak of keratitis.

The PRESIDENT said the facts which had been placed before the meeting had been very valuable, but they did not enable members to form a conclusion. These cases of interstitial keratitis of definite type in congenital syphilitics following accident or following operation were so interesting and important, that it would be a great help if members of the Society who had records and knowledge of cases would be willing to correlate those facts and bring them before the Society as a whole at a future Congress. That might enable the profession to get somewhere nearer the truth with regard to these questions.

Mr. CUNNINGHAM, in reply to Mr. Bishop Harman's remarks as to collecting cases from children's hospitals, said it was true that all his cases came from mixed clinics. He knew of Mr. Harman's pedigrees, and what he said about pregnancies fitted in with the views of Sir Jonathan Hutchinson.

In answer to Mr. Johnson Taylor's conundrum, Mr. Nettleship referred to the census of 1907, in which he found that 104 males were born to every 100 females; but the males die in excess of the females between birth and five years, so that

by the age of five—the period at which the condition under discussion counted—the sexes should be more nearly equal. In infantile iritis the disproportion was much greater, also in infantile tabes.

Mr. Holmes Spicer's cases of interstitial keratitis following injury were very interesting; his cases would work out at about 3 per cent.; his own were 2 per cent.

He had another case in mind, which he had not included here, as he regarded it as acquired. In that case the interstitial keratitis ensued soon after the injury; there were no congenital stigmata.

The remarks of Mr. Stack as to the operation for squint were important. He (the speaker) was consulted by a patient who had an old interstitial keratitis, and who wanted an operation done for squint. The Wassermann reaction was negative; should he do it?

With regard to cases in which the condition occurred in one eye only, he had notes of a number of these, the other eye not having been involved. But that was an aspect he had not entered into, as the cases were so difficult to follow up over long periods.

He felt very grateful for what the President and the Members had said; and if members cared to send him their cases of interstitial keratitis following injury, he would endeavour to collate them to the best of his ability.

4. *A case of Mooren's ulcer.*

By W. T. Holmes Spicer.

Mr. Holmes Spicer showed a case of Mooren's ulcer in which the whole of the first layers of the cornea had been destroyed. Now there was invasion of the second layer, and a few days ago perforation occurred; since then the ulceration had stopped, probably because the perforation had reduced the tension in the tissues.

The President, referring to the question of treatment of

Mooren's ulcer, said the idea recently occurred to him in connection with cases of relatively mild Mooren's ulcer to give thyroid extract. The disease appeared to be one involving a superficial portion of the cornea, and not of an infective nature. He gave it to three cases, to all of which he had had to apply repeatedly either carbolic acid or the galvano-cautery—it temporarily arrested the condition, but it always recurred—and the result of the thyroid was very satisfactory. On one occasion one of them relapsed, but the other two had gone six to twelve months without recurrence. After cauterisation the recurrences had been within six weeks. He was on the look-out for a case of more violent character, when he intended to try the thyroid on that, and see what the result would be. The dose he gave in the mild cases was about a grain twice a day. In view of degenerative changes in skin, hair, and other epiblastic tissues which were due to thyroid deficiency, he thought the use of thyroid extract in the treatment of Mooren's superficial ulcer of the cornea was worthy of further trial.

Mr. Holmes Spicer said he had tried thyroid extract in one case of Mooren's ulcer which was particularly malignant; the ulcer perforated, and the eye was lost. The man had a similar ulcer in the other eye, and was in Moorfields many months, during which time all kinds of treatment were tried. Eventually he was cured by zinc ionisation by Dr. Lewis Jones at this (St. Bartholomew's) Hospital. There were three applications of the ionisation by a zinc electrode, and the case was completely cured.

IV. DISEASES OF THE LENS.

1. *Extraction of cataract under a broad conjunctival bridge.*

By T. Harrison Butler.

There is nothing new about extraction under a conjunctival bridge. I think that it was performed by Daviel. Although I have found the operation on the whole unsatisfactory, I think that it will be useful to bring forward the reasons which appear to make it so, the more so as they have an intimate bearing upon that very important question, shall we use a conjunctival flap in extracting cataract, or shall we make our incisions purely corneal? When we make a broad conjunctival bridge, it almost immediately becomes a closed pouch, and we have in an exaggerated form the condition which is present after extraction with a wide and long flap. Any disadvantages will to some degree be shared by both.

After one or two isolated operations with a bridge I determined to perform a series. The first result was so good that I thought that I had at last hit upon the ideal method of extraction. The lens was removed on a Monday after a preliminary iridectomy, and on the Wednesday the eye had the appearance as though no operation had been done, and in a week the lady left the Hospital and had her spectacles within three weeks. I discussed the operation with Mr. Cridland and he has taken it up with enthusiasm, and will doubtless give you his views upon it. This ideal result has not been at all universal, and I propose to put before you what I have found wanting in the method.

With regard to the details of the operation:

The incision is started in the usual manner. The knife—

I use a Smith—is entered about one knife-width above the semidiameter, and the section made exactly in the corneo-scleral margin. And now comes a curious psychological fact that requires attention. When the mind is directed upon getting a good bridge, there is an unconscious tendency to think in terms of depth, and an inclination to cut too deeply. This must be resisted. The knife comes out just under the conjunctival attachment, and is then turned tangentially to the globe and the cut continued upwards till the lids or the speculum bring it to an end. Generally it will be found that a bridge almost as wide as the cornea has been obtained, and in many cases nothing else is called for. In others, however, when the eye lies deep in the socket, the bridge is not long enough. I now take a pair of squint scissors and lengthen the bridge upwards on both sides. An iris repositor is swept under the conjunctiva to tear away all fine attachments and the bridge is complete.

At first I used the scissors on one side only, and found a quite unexpected complication: The patients developed an oblique astigmatism. The rule is that the cylinder is horizontal or at most inclined up to 15 degrees, but these cases had an astigmatism up to 45 degrees. This fault disappeared when the bridge had equal sides. It was obvious that an unequal bridge caused unequal tension upon the edges of the incision, and so caused oblique healing, and oblique astigmatism.

Another advantage in having an equal opening on each side of the bridge is that the lens can escape on the side it finds most convenient and has not to be coerced to one side.

The lens is now extracted, and here we have the second disadvantage. Considerably more pressure is necessary to express the lens under a flap, because the wound will not easily gape. I regard this as a grave fault. The whole secret of successful cataract work is extreme gentleness. I dislike the word "expression." The lens should be coaxed—nay, I would almost say charmed out. If the incision be adequate, the edge of the lens will present with a minimal pressure in the right direction, and then I stab it with the

capsulotome and draw it out. Under a bridge this "charming" is not possible.

Cortex may need removal. This again is somewhat more difficult to deal with than with a wound that gapes easily. More pressure is called for.

At the end of the operation the patient is put to bed, and he will not need such careful handling as one with an open wound. This is a real advantage—one of the most valuable that the bridge gives us.

We now dress the case after 48 hours' rest. In many cases the eye is ideally quiet, but in many it looks "messy."

These bridges heal down at the edges almost immediately, and at the first dressing the incisions are difficult to make out. There is in fact a closed pouch, and, as often happens after a trephining, the aqueous spreads round the cornea, giving a general chemosis, which is harmless but does not look nice. On the whole, there is more conjunctival injection than after the orthodox operation, and for this reason all the sisters condemn it.

There is yet another somewhat serious disadvantage, and it is one which opens up the whole question of the conjunctival flap, large or small. Prof. Lundsgaard, of Copenhagen, told me at the Oxford Congress last year that the scleral section under a conjunctival flap did not heal for about 48 hours. I believe that this is true. Under a complete flap, either with or without a bridge, the wound is under isotonic conditions; there is not that almost physiological irritation which appears to be necessary for rapid healing. These operations with a broad bridge have amply confirmed this opinion. The wound is rapidly closed off from the exterior and from any risk of secondary infection, but the incision in the sclera or cornea remains open for about 48 hours. In consequence any slight trauma, such as rubbing the eye, will cause a subconjunctival hæmorrhage, and they may even occur spontaneously. The effused blood runs into the anterior chamber. In one case the Sister was away, and the nurse in charge allowed the patient to wash himself. As a result, there was a large hæmorrhage under the flap,

and the anterior chamber filled with blood. In another case some weeks after the extraction there was blood in the anterior chamber, and I could see a thick trunk of coagulated blood entering the anterior chamber from outside and expanding over the iris and the anterior layer of the capsule.

The ultimate result was perfect, but the blood took several weeks to absorb.

This late closure brings another complication in its train. Iris prolapse is more frequent, and especially after the simple operation. I have done nine simple extractions under a bridge, and there were two complete prolapses, and two more got a slight prolapse at the angles of the wound. It is true that, being entirely subconjunctival, these were harmless, and I left them alone, but it is a clumsy result and offers no advantages over the safer and similar method with iridectomy. It is well recognised that the simple method gives about 8 to 10 per cent. of iris prolapse. I have never had one when I have added a Hess peripheral iridectomy, and I now always make a basal nick in the iris when I extract without iridectomy. This addition is almost impossible under a complete bridge, and so the simple operation is at once ruled out if we make a bridge of any width.

The final disadvantage of the bridge operation is that there is a tendency to get considerably more astigmatism than after ordinary extraction. As I have already noted, if we are not careful to make the sides of the bridge equal, there will be much obliquity in the astigmatism.

If we examine the eye with Placido's disc after a week, we shall find that the figure is oblong, with rounded angles. This rapidly becomes a true oval, and in about three weeks it is a circle. After the bridge this distortion is accentuated, and recovery is slower and not always complete. In one case the refraction after two months was an 8 dioptre sphere with a 14 dioptre cylinder, and this correction gave an acuity of $\frac{6}{9}$. This high degree of astigmatism has now fallen to 11 at 175°. It is perhaps not fair to include this case, for the other lens was extracted without a bridge, and here we obtained good acuity with a 9 dioptre

cylinder. Probably there was something abnormal about the corneæ which led to excessive astigmatism. Other cases took a 5 cylinder, but the majority the ordinary 2 dioptre cylinder. There is no doubt that the broad bridge tends to excessive post-operative astigmatism.

I think that it is most useful to examine our cataract cases with Placido's disc, because it at once shows us the kind of cornea that we are dealing with, and allows us to trace the change which takes place during healing. It is the simplest test we have for irregular astigmatism. If there is any obvious departure from the circular form, it indicates an astigmatism of 6 dioptres or over.

Probably the excessive astigmatism is due to the elasticity of the bridge, which causes slight over-riding of the corneal edge of the incision.

And now with regard to the advantages :

There is no doubt that if we are dealing with a " squeezer," the bridge offers the best chance of avoiding loss of vitreous, and this is the operation of election in cases where the patient has so little self-control that it is wise to extract under general anæsthesia. In one case the patient proved to be a most determined squeezer, and it was interesting to see the wound trying in vain to gape under the patient's efforts. This was a case where I should have lost vitreous but for the bridge.

The bridge operation is best employed in conjunction with preliminary iridectomy. I do this in at least 90 per cent. of my extractions, and so I have more scope for the bridge operation.

A consideration of these patients would appear to show that if we use a conjunctival flap, it should be a small one, for a long flap, which heals at once, presents the same conditions that we get with a broad bridge, and has the same disadvantages in a modified form.

It is said that extraction under a conjunctival bridge or sub-conjunctivally is a shield against sepsis. I doubt whether this is the case. In the last 100 extractions I have lost one from iridocyclitis and two from panophthalmitis. The case of cyclitis was a bridge operation with a narrow bridge.

Sir Anderson Critchett, Bart., K.C.V.O., said that, as throughout his long career he had been a constant advocate of the combined operation, he was very glad to hear Mr. Harrison Butler say that in 90 per cent. of his cases he performed iridectomy. He asked whether Mr. Butler did the peripheral iridectomy before or after the extraction of the lens. [Mr. Butler : After.] For many years Mr. Brudenell Carter, who was exceptionally expert, did a peripheral iridectomy before attempting to extract the lens, but, in consequence of it being a procedure of such extraordinary difficulty, he abandoned it.

Personally he, Sir Anderson, could not see that the advantages of this enormous flap were so great as to at all outweigh its manifold disadvantages. He thought the ideal operation was one in which the operator got a small central flap, not carried too far back, just sufficient to help the circulation to produce rapid healing of both the section and the flap, and this, he thought, was not the case in the operation now described. And in carrying out this operation the surgeon might go so far back into the sclerotic that there was a far greater danger of rupture of the hyaloid membrane and of the loss of vitreous.

With regard to performing operations under anæsthetics, anæsthesia with nitrous oxide and oxygen could be kept up for a sufficient length of time, and sickness was usually absent. One of his most painful memories was that, before the days of cocaine, he had, within a fortnight, to enucleate two of the best extractions he ever did, on account of violent vomiting.

The operation he always aimed at was to enter the limbus, as Mr. Harrison Butler said, and emerge at the corneo-scleral margin, leaving a small central conjunctival flap.

Mr. Bernard Cridland said that when, some months ago, Mr. Harrison Butler mentioned this operation to him, it at once appealed to him, and it had been the only type of procedure he had adopted in these cases since. He had now done it in 32 cases. On the whole, to hear Mr. Butler's present views on the operation surprised him, and he had been trying to explain how Mr. Butler and he could have

arrived at such opposite conclusions on the matter. The author had summed up the operation as an unsatisfactory one, but to him, the speaker, the operation was a very satisfactory one. The difference of opinion probably arose as to the width of the flap used. He, Mr. Cridland, did not cut the flap half as wide as Mr. Butler did, and he generally began his incision lower down. He aimed to counter-puncture and keep exactly at the corneo-scleral edge. When the summit of the incision was nearly reached, then, by coming a little more into the sclera and turning the point of the knife forwards, he got to the conjunctiva and lifted it on to the knife, slid it up, and so cut a long narrow bridge. There was then little difficulty in extracting the lens.

Mr. Butler referred to the use of scissors, but only on one or two occasions had the speaker used them, for the reason that then his flap had been unusually broad. At first one would be inclined to say that a broad flap was the ideal, because it was the firmest plaster holding the cornea in its place. But it was not necessary. As long as there was a narrow tongue it acted very well in closing the wound.

Then came the difficulty of the operation, *i. e.* extracting the lens. Of his 32 cases, he had performed an involuntary iridectomy on three, the reason for that being that the anterior chamber was so shallow. The iridectomy did not disturb either him or the patients, and the results had not been bad. In one case, owing to the difficulty in extracting the lens, he did iridectomy, which he would not have done except for the difficulty of getting the lens out. In nearly all the cases he had done simple extraction, without an iridectomy, and in quite a number of the cases the lens had been coaxed out in the manner referred to by Mr. Butler. He admitted that a beginner at cataract extraction should not attempt this method; but after a little practice there was no more difficulty in this than in the ordinary method.

Mr. Butler mentioned that the sisters in the hospital condemned the operation. On the contrary, the sisters in his, the speaker's, hospital were quite pleased with it, because it allowed more licence to the patient. With the narrow flap

there were not the disadvantages of what Mr. Butler spoke of as the open wound. In one or two cases he, the speaker, had cut a broad flap, and he agreed that in those the wound had been a long time in settling down and healing. When using the narrow flap, however, it healed up nicely, and there was no surrounding œdema.

He agreed with Mr. Butler as to hæmorrhage in the anterior chamber: he had himself had two cases in which it occurred. It was the sort of contingency which might occur after any operation.

He had not had a case of prolapse of iris, but in one or two cases there had been a slight inclusion of iris. Eserine was instilled at the operation, and he thought the advantages obtained outweighed the disadvantages. He used atropine on the second day, and plenty of atropine afterwards.

With regard to astigmatism, that would occur with the broad flap, but it had not been his experience with the narrow flap. In many cataract operations it was not uncommon to find $1\frac{1}{2}$ to 2 D. of astigmatism. In a percentage of those he had done with the simple method he had not found a cylinder assisted vision in any way; that was because it was a narrow flap, and not pulling sideways, but holding up the corneal flap.

He decided he would only cover the operated eye. Patients liked that, and they were allowed up early on the third day. Usually they were allowed to return home on the eleventh or twelfth day.

With regard to the "squeezers," it gave one great satisfaction to see the futile efforts of the iron-workers, with powerful lids, doing their worst to spoil the operation. In this operation one got a quickly healing wound, and what better guarantee against sepsis could one have?

Mr. Claud Worth said he gathered from what Mr. Harrison Butler said that one advantage of the operation was in the case of the "squeezer." Most of those who had been many years on the staff of a large ophthalmic hospital could perform the operation of extraction about as well as it could be done, as regards the actual manipulation, but some obtained better results than others. He believed that the more experience

the surgeon had, the better the patients behaved. Often patients were blamed when the fault was chiefly the surgeon's.

With regard to hæmorrhage occurring afterwards, his practice was to have a small conjunctival flap. But at first, when he left the corneal incision for an incision further back, he aimed at a conjunctival flap as complete as possible. He found two disadvantages. One of them was, that almost exactly 120 hours after the operation there was liable to be hæmorrhage into the anterior chamber, when, obviously, the capillaries had developed sufficiently to bleed, and there was not firm enough union to prevent it.

Concerning post-operative astigmatism, he did not think that depended on the pull on the conjunctiva, but rather on the retention of fluid under a wide flap, delaying the healing of the wound; because when he aimed at the widest possible flap, he used to get fairly high degrees of astigmatism afterwards; and as the width of the flap was reduced, the amount of astigmatism was also reduced.

Dr. Brinton said Kaffirs were afraid of hospitals and of operations, therefore they had to be given an anæsthetic: only one in twenty was intelligent enough to allow the operation to be done without a general anæsthetic. He had done the operation Sir Anderson Critchett described, but he thought of using the operation alluded to by Mr. Cridland for Kaffirs, because they were so fidgety. Operations for cataracts in Europeans were not common in South Africa; those who could afford it preferred to come to London to get it done for nothing.

Mr. N. Bishop Harman said the margin of safety in this class of case was so small that it would be dangerous to add any complication which would make that margin still smaller. He questioned the advantages of a bridge. A conjunctival flap would stick down in less than twelve hours. What more could a bridge do? Surely the bridge of the length described could not be effective: it would allow of the escape of whatever was forced out. So that a free flap of reasonable breadth and length cut freely at the one extremity would be as effective. He was sure of the rapidity with which the conjunctiva

stuck down, because of a case he had. The operation was uneventful, and the flap was a broad one. There appeared next day a sausage-shaped swelling all along the line of the wound due to a retro-choroidal hæmorrhage; but the rapid adhesion of the flap was such that pressure only stretched it until there was a long glistening swelling along the wound, and the man's eye was saved. Why should the surgeon muddle himself up with this entanglement of a bridge for an advantage which was only problematical?

Mr. Johnson Taylor expressed his agreement with Mr. Harman; he did not see the advantage of the procedure just described. He thought conjunctiva healed in four hours.

With regard to "squeezing," the late Mr. George Lawson pointed out it was very advisable always to get the patient to keep his mouth open, and if the assistant saw that this was done, one might be sure the patient would not then be able to squeeze nearly so hard as if he had it shut.

He asked whether Mr. Butler did a preliminary iridectomy, or a peripheral one. [Mr. Butler: A preliminary one.] The speaker thought a preliminary iridectory was a great mistake, for the reason that one was doing two operations when one would suffice. Why make two bites at the cherry?

He had recently had a very rare case of hæmorrhage. He had done a simple left extraction, and everything went very well for a few days, but on the fifth day the sister said his patient had had a very bad night, and that she had had a severe pain in the left leg, causing her to compress her eyelids tightly. He opened the eye and found a hæmorrhage under the flap he had made with a hæmorrhage into the anterior chamber, looking very ugly. The left foot was cold and livid, and the posterior tibial artery was not pulsating; probably it was blocked. She had had a thrombus in that vessel, and that caused the intense pain. At first he thought there was hæmorrhage into the vitreous and retina. But it cleared up and the patient did very well and obtained very good sight. This patient some six months before had had phlebitis of the same leg.

Mr. Healy, speaking of "squeezers," said that iron-workers

and those who worked in similar trades got a big development of the orbicularis ; and he would rather operate on a hysterical woman than on many of these iron-workers. When operating on these workers, unless one was very careful, they would lose vitreous with great ease, owing to sudden and unwarranted rat-trap contractions.

He had two trials of Mr. Cridland's operation. In one it was a success ; in the other there was loss of much vitreous and prolapse of iris. The latter might have been due to the fact that he tried to do a complete iridectomy at the time. It was very difficult, and it was not a safe course to do complete iridectomy at the time of this operation.

Mr. GREENE (Norwich) said Prof. MacHardy always made his cataract patients not only keep their mouths open, but breathe through the mouth the whole time ; and he usually finished up by saying, "Don't talk, and don't thank God."

Mr. TRAQUAIR remarked that a good deal had been said about "squeezing." There was an efficient method of preventing that, which in Edinburgh had been found so effective that it was, in one clinic, practically a routine. It consisted in paralysing the orbicularis by injections of novocaine—a method advocated some years ago, and apparently a common practice on the continent. Injections of 2 per cent. novocaine made about the upper, lower and outer aspects of the orbicularis produced a paralysis so complete that in about ten minutes the patient could not shut his eye. After the eye had been operated upon it remained open for a short time, but by keeping a pad on for 24 hours no bad results ensued. One of the Edinburgh surgeons used this method as a routine, and he, the speaker, did so when he had reason to think a particular patient would be a "squeezer."

There was another small matter of detail with regard to the possibility of doing a peripheral iridectomy underneath the bridge. He had never seen it done underneath a bridge, but when he was in Innsbruck before the war, the late Prof. St. Bernheimer told him he always did a peripheral

iridectomy, passing his forceps and scissors beneath the flap and cutting the iris without withdrawing it beyond the margin of the incision. If that was possible, it should also be possible to do an iridectomy beneath Mr. Cridland's bridge. He did not mention this to give an opinion on the advantages or otherwise of the operation, but to help the performance of it.

Mr. Harrison Butler (in reply) said that it had been most valuable to hear Sir Anderson Critchett's opinion upon these questions, for all valued his unique experience in cataract extraction.

With regard to anæsthesia, he had extracted four or five times under general anæsthesia, and in all with favourable results. The time necessary was so short that, provided the anæsthetist knew his job, there was not likely to be any vomiting. He insisted upon ether because he had had personal experience of many deaths from chloroform, although none of them had been his own cases. His experience with ether had been so gratifying that when he knew that a man was a bad squeezer he would not hesitate to put him under general anæsthesia, but it must be profound.

In answer to Mr. Cridland, he thought that his better results were due to the fact that he made a narrow bridge which left part of the incision exposed. It was not at all easy to cut a bridge of any desired width. If one started with the idea of a narrow bridge, it might easily happen that the result was on broad lines.

He considered that if there was any injury to the eye after operation, then the bridge or large flap was a great protection. He had seen many traumatic sub-conjunctival hæmorrhages which with a corneal section would have been external ones with great risk of infection.

He met with but few squeezers in a well-managed clinic, but in a small hospital with ill-trained sisters in the ophthalmic sense they were much commoner: in fact, he found that it was quite impossible to get a good series of extractions in one of his hospitals where there were few extractions, and the staff could not obtain the necessary training. Favourable

statistics could be obtained only in an eye hospital or in a general hospital with an eye department, under a properly trained sister, where there was enough material to train the staff.

In answer to Mr. Bishop Harman, he would say most emphatically that in spite of all that Mr. Harman had said a good bridge was most efficient in circumventing a squeezer.

He always tried to remember to ask the patient to keep his mouth open, and he put cocaine into both eyes.

He would not be drawn into a discussion upon the advantage of preliminary iridectomy, but he was quite willing to defend it at any subsequent congress. His operation would be one of two: He would perform preliminary iridectomy or he would go the whole hog and adopt simple extraction with a peripheral iris nick.

(The President pointed out that some speakers used the word "flap" when they meant "bridge.")

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2. *Greyish-white spots of the anterior lens capsule after abscission of iris for traumatic prolapse.*

By Basil Graves.

Some fifteen months ago whilst resident in Moorfields Hospital I noticed a feature about the anterior lens capsule of a case which I was discharging about twenty-four days after I had done an abscission for traumatic prolapse of the iris. Incidentally, although I did not record it in the notes, this rather long period before the patient's discharge from hospital was doubtless accounted for by the fact that the traumatic wound encroached on the ciliary region. Some two months later I remarked a similar condition in a similar case when examining him preparatory to discharge on the eleventh day after I had abscised some prolapsed iris. The first case was under the care of Mr. Basil Lang, the second under Sir John Parsons. I have with me copies of the

in-patient notes which I made on these two cases: both subsequently attained $\frac{6}{12}$ vision with the affected eye. Later, when the recollection of these two cases had rather lapsed, and when I was not specially seeking for this condition, a third such case bearing a similar history recalled the condition to my mind. I have subsequently met with three more cases, making six in all; the last two, originally under the care of Mr. Treacher Collins, and now of Mr. P. G. Doyne, are coming up this afternoon to the clinical meeting. I am thus able to give the precise history of four of these six patients; the in-patient notes on the remaining two are at present in the hands of the bookbinder, and, other than to say from memory that they bore a similar surgical history, I shall not instance them further.

The feature of these cases is some fine thin unpigmented greyish-white discrete spots of the anterior lens capsule. I can state fairly definite negative information as to what I feel they are not, but it is a little difficult to be positive as to what they are. I take them to be a localised capsular proliferation. They were detected in the first instance by the usual × 10 monocular pocket-loupe with oblique focal illumination. They are naturally better studied with the binocular eye-microscope, in conjunction with an intensive illumination. I have been experimenting to obtain an intensified form of contact illumination, and for the moment have used for the purpose a "pointolite" source from which parallel rays are brought into an internally-reflecting silvered glass rod. The terminal edge of this rod is ground into a parabolic bevel and a high concentration of rays is brought to bear upon the terminal contact face. For examination of the lens capsule contact is applied, according to the effect required, to the cornea or to the limbal region, but in the latter case the diameter of the rod must not exceed the limits covered by the ciliary body, otherwise a vivid red reflex forms the background owing to the very powerful trans-scleral projection possible with this appliance. In two of the cases under discussion, thus examined with high magnification, some of the spots revealed very fine short radiating filamentary

lines tending to recede slightly backwards from the lens capsule. Many of these lines are connected with or terminate in fine stellate branchings. The precise hue of these spots varies, as would be expected, mainly with the contrast phenomena dependent on the optical conditions of their illumination. With a strictly limited intensive focal beam, such as that of the Gullstrand slit-lamp, directed very obliquely so as to produce a minimal illumination of the underlying lens substance, these spots appear very white. Ordinary widespread oblique illumination, in which some light penetrates and illuminates the general lens substance, reveals these spots, as might be expected, in a duller grey. Finally, retinoscopic illumination shows that these spots do not interrupt the ordinary red reflex, which fact is definite evidence of their extreme thinness, and also of the fact that uveal pigment does not enter into their composition. In two of the cases a few fair-sized spots of uveal pigment or pigmented lymph have in addition, and independently, been present on the lens capsule; in the other cases they were absent.

Although the spots in the two cases which I hope to show you to-day are more or less paracentral on the lens capsule, the other cases which I have seen reveal these spots as by no means restricted or specially related to the pupillary region of the capsule. Their mutual grouping, rather close, tends usually to be linear. The total number of the spots in each case varies usually from two to about ten.

If eyes be studied under high magnification, various minute peculiarities of developmental origin are frequently discoverable in the region of the anterior lens capsule. The white spots I am describing do not resemble these.

It may be asked whether they may not be inflammatory deposits, or a sequel in effect to the preliminary presence of such deposits. With a view to studying the destiny of minute inflammatory deposits and their ultimate effect on the adjacent lens capsule, I have for over two years been closely following up some cases of former iritis in which minute inflammatory remnants had been left on the anterior lens-capsule. Without labouring the features of these latter now, I may say that the

spots which I am discussing do not resemble the after-effects of such inflammatory deposits.

Possibly a deposit of fibrin was a suggestion by Mr. Treacher Collins. In four or five cases of injured eyes examined on their prompt admission to hospital I have seen a formed material in the anterior chamber, chiefly disposed as flocculent untinted semi-translucent strands, appearing to be of a rather more tangible consistency than that merely of fresh lymph. One of these cases was a concussion injury without perforation. All had more or less hyphæma, but the strands themselves were colourless. On first sight this condition immediately suggests a torn lens capsule, and discrimination may be difficult. These cases are rare; the condition is seen when the patient is admitted shortly after his accident; in the cases which I have seen it had disappeared by the second or third day after admission, and before the entire disappearance of the associated hyphæma. One such strand running forward and upwards through the pupil on to the front of the upper part of the iris was present in Sir John Parsons' case (J. M—), which I have quoted as subsequently revealing white specks of the lens capsule, and it is illustrated in the notes which I made on the admission of this patient. This strand, I remember, had disappeared when the eye was examined the day after I had abscised the prolapse, and the lens capsule was then seen to be intact. I may say that for certain reasons I limited the abscission only to the extreme inferior peripheral region of the iris, and irrigation was not used in this case, therefore it was natural to look for the presence of this strand when dressing the case next morning. I suggest this rather rare and transient condition in the anterior chamber to be probably freshly precipitated blood-fibrin devoid of corpuscles, which subsequently undergoes absorption. Recent examination of this case, *i. e.* a year after the accident, still reveals two white spots on the anterior lens capsule recorded as being present on his discharge from hospital, and as in this case the spots are whiter and denser than those seen in the other cases of capsular spots which I have on record, I take it that in this one case the explanation

of some hæmatogenous deposit taking part in the formation of the spots is not inadmissible ; but the appearance of the spots in the remaining five cases, two of whom should be available for examination this afternoon, to my mind renders it unlikely that they are due to a deposition of fibrin.

Their position precludes the suggestion that they may have been due to contact of the teeth of iris-forceps with the lens capsule. In three of the cases I know that no instrument was inserted into the anterior chamber, and that the pillars of the iris after abscission were replaced by irrigating, and not by the use of a repositor.

I am not aware of these white spots being present after ordinary non-perforating concussion injuries with or without hyphæma, and up to a few days ago I should have said that I have not seen a similar occurrence after ordinary iridectomies on non-traumatic cases, or for that matter as a sequel to any of the every-day intra-ocular operations. It happened that two days ago I was examining a case which I had trephined for chronic primary glaucoma some six weeks previously, and I came upon some grey spots rather similar to those in the traumatic cases described—at least, they are rather similar under ordinary oblique illumination.

I have not had an opportunity to examine this case in the short time since then under intensive illumination. However, a chance pigment-speck deposited near by on the front of the lens capsule affords a standard for stereoscopic level, and it is quite apparent that these grey spots of this trephined case are in the lens substance immediately subjacent to the capsule. This case therefore differs from the preceding ones in that the spots lie rather deeper. The case was an ordinary one except that the trephine hole, being made up and out over a region of the iris which revealed certain features of interest, I made a complete iridectomy in order to obtain that part of the iris for histological section. When a deliberate complete iridectomy is done with sclero-corneal trephining the pillars spring back, and it is unnecessary to introduce any repositor, and it is often unnecessary even to stroke the cornea in order to replace the iris-pillars. This case is also coming up this

afternoon, and I mention these details to intimate that the lens was not touched by any instrument or influenced by manipulation over the cornea.

In this glaucoma-trephine case, in addition to these spots, which are not far from the centre of the lens capsule, there is also near the centre a faint gossamer-like diaphanous opacity of the subcapsular lens substance. It does not interfere with the corrected vision being $\frac{6}{6}$. I used at one time to think that this small central opacification was a sequel to prolonged contact of the lens with the back of the cornea following undue delay in the re-formation of the anterior chamber after trephining, but it so happens that in this particular case a definite anterior chamber was present when the case was dressed twenty-four hours after the operation, and was not observed to collapse thereafter.

It remains to intimate the nature of the accident which befel the four cases of traumatic prolapse whose histories are accessible at the present moment:

H. G—, æt. 28 years, struck in L.E. by a piece of glass, which he says measured about $\frac{3}{4}$ in. square, through the bursting of a boiler-gauge glass. A small radial limbal wound, down and in, with minimal prolapse of the periphery of the iris only just sufficient to make the pupil slightly oval. A trace of hyphæma. Ultimate vision, $\frac{6}{12}$.

J. M—, æt. 26 years, hit in R.E. by large piece of steel, which he states he saw fall to the ground, while lathe-turning. An extensive crescentic corneal wound from temporal side of mid-cornea, running down and in to limbus, in spite of which, however, there was only a minimal prolapse of iris. Ultimate vision, $\frac{6}{12}$.

L. G—, æt. 30 years, struck in R.E. by piece of glass from a window shattered by an air-gun pellet. Ultimate vision $\frac{6}{6}$ in injured eye. This case will be here this afternoon.

F. J. T—, æt. 18 years, struck by a piece of steel thrown by another boy. An extensive flap-wound of the lower part of the cornea. This is the second case coming up this afternoon for examination.

In conclusion, the similarity of the histories of these few

cases I have quoted has led me to record a feature which perhaps in itself is small and of no particular importance. Further observation will reveal whether or not it is justifiable to say that the condition is specifically related to this class of traumatic cases.

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3. *Difficulties in removing hard cataract.*

By G. F. Alexander.

1. *In sterilising the conjunctiva.*—With irrigation as usually practised the fluid never reaches the upper fornix, as even if the upper lid is raised by the speculum a fold of loose conjunctiva so encircles the globe in front of the equator as to shut off the flow from the fornix. The difficulty can only be overcome by passing a nozzle such as I show to the upper limit of the fornix and sweeping over it from side to side before introducing the speculum.

2. *Muscular.*—(A) Uncontrollable spasm of the orbicularis apt in the presence of the speculum to cause dangerous pressure on the eye. That this is difficult to combat is evident from the number of the devices for doing so, and such a statement as that by Mr. Maddox at the meeting of the British Medical Association in 1914: "The dozens of eyes that are spoiled annually in this country by sudden squeezes may well make us cast about for some way of making all squeezes ineffective." When the lids are separated by the speculum they lie back on the eyeball, so that the fibres of the pars palpebralis encircle it behind the cornea sphincter-like, and therefore in the effort to close the lids compress it equatorially.

(B) The effort to roll up the eye against the resistance of the forceps, which causes the section to gape and thus tends to cause loss of vitreous, necessitating the eye being released immediately the section is completed. Obviously the above remark of Maddox is equally applicable to unwanted movements of the eye as well as to squeezing. Thus for this

operation, requiring above all others the most delicate manipulation, we are left at the mercy of the patient's self-control.

Cocaine does not prevent spasm of the orbicularis or rolling up of the eye even in the absence of all pain, as either may take place from its mere anticipation, and it seems that in chloroform anæsthesia we have the only absolutely reliable means of preventing both.

The disadvantages of chloroform I find stated as follows: –

1. There is a tendency for the eye to roll up; but this I have found either absent or so slight that it can be overcome by a light pull of the forceps, and once pulled down for the section the eye may remain sufficiently low for the subsequent steps of the operation, and, if not, can be held so by a grip below the cornea in perfect safety.

2. There is a danger of vomiting forcing open the section and causing prolapse of the iris, loss of vitreous, and intraocular hæmorrhages of all degrees of severity; but so little chloroform is required that often there is no vomiting, and even if it does follow, as long as the section holds no harm results, and I feel justified in claiming that this is secured by the dressing I advocated at the meeting of this Society in 1914, viz. a "fluid pad" applied for the first day with slight pressure by the bandage, *i. e.* little more than is required to keep the dressing in position, as to which it is interesting to find this identical dressing recommended by the late Colonel Maynard, of Calcutta, in his manual published in 1920, though he does not allude to the benefit derived from pressure, and though my remarks drew the reply that all pressure should be avoided as causing over-riding of the scleral by the corneal flap, this is not the case with pressure applied evenly over the cornea, and with pressure applied otherwise the reverse tends to take place, *i. e.* the corneal flap tends to be turned down from the scleral so that the section gapes.

I was led to rely on this dressing after having three cases of cocaine anæsthesia in which vomiting from biliousness followed the night of the operation without affecting the section in the least, and what seemed the "experimentum

crucis " being supplied by the last of these—a totally deaf person in whom I had to do a vectis extraction who was sick with severe straining three times.

In the case of a deaf person, a highly nervous person, one whose other eye is useless for vision, and when cocaine fails to act satisfactorily, chloroform should surely be given, and I am in hopes that its use may pave the way for the intra-capsular removal of the lens with greater immunity from loss of vitreous than is at present the case.

3. Inability of the operator to control the eye. The forceps applied close to the cornea may fail to grip, and even if it holds the conjunctiva at times stretches so that control is lost, and not unfrequently in old people the forceps under even a light pull tears out or if holding strips a flap of conjunctiva from the sclerotic. A grip of any tendon may from the thickness of the conjunctiva or the eye being deeply set in the orbit prove far from easy, and with cocaine one of the internal rectus tendon may not give perfect control and one of the superior rectus tendon may prove painful.

The difficulty lies in the teeth of most forceps being at right angles to the blades, so that they grasp only the conjunctiva, and then only by a fold so broad that it tends to be drawn over the limbus, and consequently cut by the Graefe knife at the counter-puncture, and is overcome by so inclining the teeth that they can bite into the episcleral tissue, as I found in the Birmingham pattern made by Messrs. Down; but I find still better control given by passing the sharp hook I show made by Weiss into the sclerotic by aid of Batten's fixation fork, this having the advantages over the forceps of being more easily transferred to an assistant, and of not drawing the conjunctiva over the limbus, so that a grip may be made inside the cornea without this membrane being cut.

4. As to the size of the section:—Applying the terms " limbus " to the outer or visible cornea-sclerotic junction, " outlet " to the chord or base of the section of the inner surface of the cornea, and " altitude " to the distance of the outlet above the centre of the limbus, the latter is most easily judged of from its relation to the pupil and the vertex

of the cornea. Taking the diameter of the limbus as 11·5 mm., and the radial thickness of the cornea as 1 mm., as the thickness of the wall in a limbal section is 1·5 mm. it is easily shown that in a section taking in one-third of the limbus the outlet is 6·3 mm. or if the puncture is made with the knife directed slightly downwards, 7 mm.

Now, as I found from a number of measurements in Col. Elliot's clinic in Madras, the average diameter of the disc delivered after capsulotomy, which consists of the nucleus surrounded in all cases but Morgagnian by a variable amount of specially adhesive cortex, averages 6·7 mm.; hence, as this cortex is mouldable, the above section amply suffices for the delivery of the average nucleus. It may therefore be taken as the standard section, and as if it proves too small there is a way out of the difficulty without enlarging it, it need never be exceeded unless to express the lens in its capsule. This section is obtained by giving it an altitude midway between the centre of the pupil and the vertex of the cornea, and therefore nearly 3 mm. (actually $1\frac{1\cdot5}{4} = 2\cdot87$ mm.) above the former and below the latter, in which connection I desire to draw attention to a widely followed error on the part of Prof. Meller of Vienna leading to the section being made larger than need be, viz. with the aim of taking in one-third of the limbus he directs for this the puncture to be 1 mm. above the horizontal diameter of the cornea, whereas it should be 2 mm. higher than this.

5. In making the section: (1) The advice to hold the Graefe knife lightly should surely be amended, as though the pressure by the blade must necessarily be minimal, for the command of finely graduated movements the handle should naturally be held firmly. (2) Pointing 1 mm. in front of the limbus in order to counter-puncture in it, as is so often advised, is apt to restrict the outlet inwardly; ·5 mm. is the correct distance, as the image of the knife is advanced by the refraction of the cornea to only this extent. (3) The knife I show made by Weiss possesses for the shorter sections in the limbus the advantages of the Beer knife, viz. completion by simply extending the digits, perfect regularity, and freedom

from the risk of its edge cutting the iris, while as at 6 mm. from its point it is only 1·5 mm. broad it can be made to clear the iris in a shallow anterior chamber or do capsulotomy as easily as a narrow Graefe knife: it thus combines the chief advantages of the two knives. (4) Occasionally there is with any knife a difficulty in completing the section in the limbus in the first thrust, due to the eye, if fixed below the cornea, which gives it the fullest downward rotation, being free to rotate in under the thrust, and doing so to an extent which renders sawing necessary; if, however, an assistant holds the eye down by the hook or forceps, and opposes the knife with the fixation fork just within the cornea below the counter-puncture, it renders the section easy even in the case of a deep-set eye and narrow palpebral aperture: the points of the fork merely catch in the sub-conjunctival tissue.

6. The prevention of after-cataract:—From observations on animal's eyes it is evident that (*a*) in capsulotomy, however we devise the incision, it is altogether deranged first by the cystitome itself, and second by the passage of the lens. Even if the cystitome is perfectly sharp and drawn over the whole length of the vertical meridian of the lens, the capsule frequently so curls up before its advancing edge that the incision is limited upwards, and this is frequently accompanied by lateral tearing, either transversely as if we had made a transverse incision, which by leaving a pocket above explains the difficulty in the lens being made to present and its occasional delivery by tumbling, *i. e.* its rotation so that its lower border comes first through the section, or upwards and outwards, in which case we are left with a triangular flap pointing down underlying the pupil. It is impossible to cut out a pattern. The lens naturally from its shape tends to tear the capsule horizontally, and may thus convert even a limited vertical capsulotomy into a horizontal one; but it frequently tears in several directions, so that tags are formed. (*b*) In capsulectomy with the forceps (1) such force is at times required that the hyaloid is apt to be torn and thus vitreous to be lost; but if resistance is encountered, this

risk seems diminished by removing the capsule by a twisting movement, as this distributes the strain evenly over the zonule, whereas tugging movements throw it on one quadrant at a time. (2) Occasionally the part of the capsule detached is so small that tags result from the passage of the lens, and even if a large area is detached only part of it may come away in the forceps; hence we should aim at bringing away a large area by passing forceps with long teeth-bearing blades under the lower quadrant of the iris and separating the blades widely. In either case we should under strong illumination look for tags floated up on irrigating and pull them away by iris forceps, or preferably a similar form without the sharp points, and with the inner surfaces of the curves roughened, as I show.

7. (*a*) Failure of the lens to present:—Apart from insufficient capsulotomy, this may be due to lack of what in an eye of normal intra-ocular pressure and uninfluenced by muscularity is termed vitreous tension—a term I consider erroneous, and should be replaced by scleral tension, as, the vitreous being passive, the forward thrust of the lens is due to the elasticity of the sclera, and, when this is wanting, as it is in high myopia from atrophy of the sclera, which may be so great as to lead to its falling in, the lens refuses to leave the fossa patellaris under pressure exerted on the cornea, and can only be got to do so by pressing near to the equator of the globe, while instead of there being a tendency to loss of vitreous the reverse is the case. The view that the sclerotic is elastic is contrary to what I have frequently heard expressed in this society, but I hold it is proved by a puncture into the anterior chamber of the eye being followed by forcible expulsion of the aqueous, for as the blood is not in continuity with the aqueous this can only be explained by the elasticity of the sclera.

(*b*) Failure to express the lens when presenting:—We should first try side-to-side movements of the curette or ball-point of a strabismus hook over the cornea, as if the outlet is limited on one side only this may roll the nucleus over the resisting edge of the inner section, and if this fails, we should, while

supporting the nucleus below and on the right, thrust deeply into it from the left the nucleus hook I show and extract it, and if in doing so it splits in two as the hook brings away the upper half the lower is then easily expressed. The Graefe knife held flat or a flat-headed discission needle answers equally well. The retention of a little extra cortex which may result need not trouble an operator using the irrigator, as it is not attached to the capsule.

8. The irrigator I show is very portable, can be held in one hand by the operator, is its own sterilising vessel and nozzle cup, requires no assembling, and gives a flow by syphonage automatically, and thus seems the acme of simplicity, while costing only a few pence. It consists of a half-pint Florence flask, reaching to the bottom of which is a glass tube of 4 mm. bore with a horse-shoe bend at the mouth of the flask, to this is attached a rubber tube of 3 mm. bore carrying the nozzle. If the flask is filled to within half an inch of the neck and the nozzle immersed half its length in the fluid, and the flask gently boiled for 15 minutes, as steam or boiling lotion circulates in the tubes, the interior of the rubber tube cannot fail to be sterilised, and if, when required, the rubber tube is gripped by the finger and thumb at the mouth of the flask and withdrawn, a head of fluid is obtained.

As filaments of flock, etc., gain access to all fluids kept in bottles and confervoid growth takes place in saline solutions, as these particles, though sterile, may act as mechanical irritants, the fluid should immediately before use be passed through a Berkefeld filter.

Mr. Claud Worth said he did not know whether any other members made the incision as small as Mr. Alexander did. Before his own practice crystallised he often regretted having made the incision too small, but he never remembered having cause to regret too large an incision.

V. DISEASES OF THE RETINA AND OPTIC NERVE.

1. *A congenital anomaly of the optic nerve.*

By Frank Juler and Ida C. Mann.

(With Plates I and II.)

In a paper published in 1909 the late George Coats (1) brought forward a theory (already propounded by Monthus and Opin) that a certain group of congenital anomalies of the eye might be explained by supposing that any part of the secondary optic vesicle and optic stalk may occasionally differentiate, perfectly or imperfectly, into any of the types of tissue normally formed from the optic outgrowth, namely, pigmented epithelium, unpigmented epithelium, retina or neuroglia. If this is so, it should be possible to find examples of these four kinds of tissue occurring in abnormal situations in the eye or optic nerve. Coats, in his original paper, tabulated the possibilities for the four sites, iris, ciliary body, optic nerve and retina, and quoted examples of some of them from recorded cases.

In the present paper we are describing a case which appears to be explicable by the above theory, and which we should class as an anomaly of differentiation of the optic stalk.

In the normal course of development the optic stalk, as well as the optic vesicle, is invaginated by the fœtal fissure, so that two layers, an inner and an outer, are formed. The *inner* layer of the optic cup in its posterior part proliferates, becomes several cells thick and differentiates to form the retina. The inner layer of the *stalk*, with which it is directly continuous, does not normally make any attempt to form

retinal tissue, but is invaded by issuing nerve-fibres, and finally helps to form the neuroglial supporting tissue of the optic nerve. The *outer* layer of the optic cup remains only one cell thick, develops pigment and forms the hexagonal pigment epithelium of the retina; the outer layer of the optic *stalk* normally shows no signs of pigmentation, and usually disappears entirely, possibly taking part in the formation of neuroglia or blending with and becoming lost in the sheath of the nerve.

In the case under consideration it seems likely that differentiation of the inner and outer layers of the optic cup did not stop at the point of insertion of the stalk into the cup, but involved also the inner and outer layers of the stalk for some little distance, the outer layer becoming pigmented and the inner proliferating in an attempt to form retina, which is, however, atypical. There are some minor abnormalities of the choroid, retina and sclerotic associated with this.

Coats himself cites two cases of formation of retina in the optic nerve. They occurred in the two eyes of an anencephalic child, and had been previously classified by him as small colobomata of the nerve-sheath (2). There were no nerve-fibres, and associated with the abortive retinal elements in the nerve were layers of irregular pigment formation. Coats suggested that some cases of pigmented crater-like holes at the optic disc might be explained in the same manner.

Monthus and Opin (3) have described a case with excessive pigmentation surrounding an optic stalk which contained no nerve fibres. It occurred in a child born with a cystic condition of the occipital lobes, and also double hare-lip, cleft palate and polydactyly. It was associated with a very small and malformed eye (measuring only 4 mm. across), and there appeared to have been incomplete invagination of the primary optic vesicle.

Pigmentation of the nerve occurs normally in bat embryos (Nussbaum). Seefelder (4) reports the findings in a 5½-day chick embryo with unilateral cryptophthalmia. The rudimentary eye consisted of a stem of pigmented epithelium connected with the brain, which ended below in finger-like

processes in the position of the eyeball, representing the primary optic vesicle; the retina had not developed, and there was no trace of a lens. In its course this epithelial layer showed certain clear spaces, which at first appeared to be vacuoles, but which were really filled with nerve-fibres. These fibres at some distance from the brain formed a bundle in the solid stem, which towards its central end lost its pigment.

Buchanan (5) described a small bundle of medullated nerve-fibres coursing between the retina and pigment epithelium to the disc margin. He explained it by a folding of the layers of the secondary optic vesicle, but in the discussion Treacher Collins and Mayou suggested that it might be due to an outgrowth of fibres from the outer layer of the secondary vesicle.

Clinical history.—A child, R. R—, aged 9 months, was admitted to the East London Hospital for Children under the care of Mr. Bowen in 1909, and died from a large sarcoma of the left middle ear within two months of admission (6).

The notes record that instruments had been used at birth, and the head had been injured thereby, causing a wound on the left side. On admission there was a swelling on the left side of the neck opposite the angle of the jaw, and aural polypi were present. The child was dull and apathetic, almost blind, with a slow wide nystagmus, but no head-nodding; the fundi showed "atrophic choroiditis."

Ophthalmoscopic appearances.—The late Mr. Ilbert Hancock and one of us (F. J.) examined the eyes, but unfortunately the rough sketches made by the former have not been found, nor are any notes made to his dictation obtainable. The following are the only details of the ophthalmoscopic appearance that can be remembered.

The site of the left optic papilla was occupied by a black mass, no details of which could be made out. Retinal vessels of normal appearance disappeared into the black area. Parts of both fundi showed irregular patches of choroidal atrophy without heaping up of pigment.

Macroscopic examination.—Post-mortem examination (though incomplete) revealed no intracranial growth.

The left eye was removed and preserved in formalin for some time and shrank considerably in its anterior part; the cut end of the nerve showed a thin black ring surrounding it, apparently just beneath the sheath.

The globe was divided in the vertical antero-posterior plane, and was cut in serial sections, which were, for the most part, stained with hæmatoxylin and eosin.

Owing to the collapse of the globe, exact measurements are not possible, but roughly the antero-posterior diameter is 19 mm.

The lens is apparently normal, but has fallen to the bottom of the vitreous cavity, which is well formed; there is no detachment of the retina.

Microscopic examination.—The anterior part of the globe is normal except for a slight cystic change in the posterior pigment layers of the iris.

There is no ectasia of the globe; the sclera is normal, except in the region of the nerve, where, instead of ending sharply at the edge of the scleral foramen, it curves gradually backwards to become continuous with the fibrous tissue of the nerve-sheath, with which it blends imperceptibly. This is seen in Pl. I, figs. 1 and 2.

The choroid, which contains pigment, appears to be thinner than normal, and it is difficult to distinguish its constituent layers. This may in part be due to the formalin fixation. At the edge of the nerve, the choroid, instead of ending sharply as usual, is curved like the sclera backwards along the nerve, but only extends a short distance, becoming very thin and soon fading away (Pl. I, fig. 2, A).

The retina is well formed in its outer layers: the rod and cone layer is well marked (considering the formalin fixation), and both nuclear layers are well defined and regular. The ganglion cell layer is present, but the cells are fewer in number than usual. The nerve-fibre layer is very defective. It is everywhere thinner than normal, and the increase in thickness which is usually seen as one approaches the optic disc is not at all well marked. The retinal vessels, which usually appear deeply embedded in this layer, are more superficially

PLATE I.

Illustrates Mr. Frank Juler's and Miss Ida C. Mann's paper on A Congenital Anomaly of the Optic Nerve (p. 87).

FIG. 1 is a vertical section near the nasal edge of the nerve-head.

FIG. 2 passes through the nerve on the nasal side.

The retina is seen to end gradually, its various layers fading off into the tissue of the nerve, which in fig. 2 contains numerous corpora amylacea. Note the poor development of the nerve-fibre and ganglion cell layers, the rosette-like formations on the nerve-head, the pigment layer continuous around the scleral curve with the pigmented sheath, the amorphous layer (B) internal to this sheath, its contained pigment proliferations (T), and the obliquely cut nerve (N) posteriorly, which in fig. 2 is less clearly shown, but which occupies the whole of the posterior portion of the nerve. (A) points to the choroid as it fades off around the scleral curve. There is no distinct lamina cribrosa.

Fig. 1.

Fig. 2

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PLATE II.

Illustrates Mr. Frank Juler's and Miss Ida C. Mann's paper on A Congenital Anomaly of the Optic Nerve (p. 87).

Fig. 3 passes through the temporal side of the nerve, and shows nerve-fibres running backwards from the front of the retina; large veins are also visible. The general arrangement of the pigmented sheath and its continuity with the pigment layer of the retina are similar to that in fig. 2.

Fig. 4 is taken from a section close to the temporal margin. The nerve contains a few nerve-fibres and some strands of fibrous tissue, one bundle of which is especially well marked and runs forwards from the cut end to the depression on the nerve-head (? mesoderm).

Fig. 3.

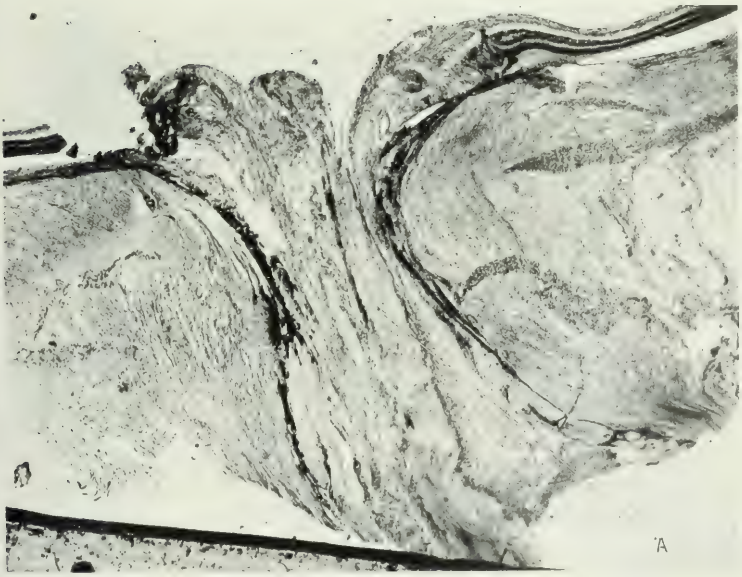

Fig. 4.

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placed, and in some situations almost appear to stand out from the surface in a way suggestive of the retina of an anencephalic fœtus. The supporting tissue of the retina is very well developed ; the fibres of Müller are readily recognised and form a normal internal limiting membrane.

The *pigment epithelium* within the globe is for the most part normal, and in places a lamina vitrea can be made out. In one area there is a zone in which the epithelium is unpigmented ; it is not extensive, and occurs above the disc. There are two or three oval collections of cells, in connection with the epithelial layer in this region, which project into the rod and cone layer, and whose nuclei exactly resemble those of the epithelial layer. Though most of these cells are unpigmented, a few contain some brown granules, and they are obviously an irregularity of the pigment epithelium. The arrangement of the pigment at the disc will be described below.

In places there is a thin amorphous layer of pigmented granular material between the pigment epithelium and the retina, which may, however, have been produced by post-mortem change or fixation.

The optic nerve and papilla.—The chief abnormality lies in the condition of the optic nerve and adjacent retina with its pigmented epithelium. The dark ring noted macroscopically is found to be due to an extension of the pigment epithelium of the retina backwards along the optic nerve. This extension has involved the whole of that portion of the nerve seen in the sections, with the exception of a small area at the posterior end in the upper nasal quadrant (Pls. I and II).

In the lower temporal quadrant there can be seen in a few sections a narrow longitudinal gap in the pigmented epithelium, which appears to be slightly invaginated along its margins, the incurved portions being continuous with the tissue inside the nerve. It is possible that this narrow gap may represent some remnant of the fœtal fissure in the nerve, and this opinion is strengthened by the fact that fibrous tissue (mesoderm) enters this gap and can be traced in continuity with the fibrous tissue in the nerve. A small

blood-vessel, apparently an arteriole, also enters the nerve through this gap in one section. It passes into the fibrous tissue in the nerve, but it is impossible to say whether it has any connection with the central vessels. There are, however, also other vascular communications by capillaries between the neural and scleral vessels.

The edges of the scleral foramen anteriorly are rounded off as the pigment epithelium courses backwards, the pigment epithelium of the retina being directly continuous along this curve with that around the nerve. The arrangement of the pigment along the nerve, however, is not perfectly regular. In some places the cells and their nuclei are exactly similar to those of normal pigment epithelium, while in others they tend to become four- or five-sided instead of hexagonal. The arrangement of the pigment is not in a continuous single layer; in some places there are small gaps (Pl. II, fig. 3); in other parts the layer is more than one cell thick (Pl. I, figs. 1 and 2); in other parts, again, especially on the nasal side, and near the papilla, the layer has proliferated into numerous processes, showing a somewhat alveolar and racemose arrangement, which penetrate into the adjacent tissue towards the interior of the nerve (Pl. I, fig. 1).

This tissue which lines the inner surface of the pigment layer on the nerve is peculiar. It forms a layer, stained pink in the sections, which surrounds the nerve beneath the pigment, being of varying thickness, very narrow, especially above, in the central sections, and thicker on the nasal side. Posteriorly it blends with the dural sheath beyond the pigment in the upper nasal quadrant, and is continuous with this sheath through small gaps in the pigment at many points. It resembles fibrous tissue, but in parts is very poor in nuclei (Pl. I, fig. 2), appearing almost homogeneous, though faint fibrillations can generally be made out; in other places it is more definitely fibrous with spindle-shaped nuclei. In it, especially on the nasal side, ramify the proliferations of the pigment epithelium (which resemble the formations seen in cyclitic membranes), and it appears to be connected intimately with the pigmented layer. In places it appears as if enclosed

between two layers of pigment (Pl. II, fig. 3). It contains no blood-vessels. Its nature is obscure. It may be ectodermal in origin, and derived from the abnormal pigment epithelium along the nerve, in which case it can be looked on as related to neuroglia, or it may be a part of the dural sheath of the nerve (mesoderm), which has been invaded by patches of pigment epithelium derived from cell remains of the outer layer of the optic stalk which have persisted more or less scattered among the fibres of the sheath.

The nerve enters the scleral foramen from above and the nasal side, and is seen in oblique section in some of the micro-photographs (Pl. I, fig. 1).

The interior of the nerve is highly abnormal. There are very few nerve-fibres, and there is no attempt in the anterior part at arrangement into bundles separated by fibrous septa. The posterior part of the nerve appears bent, and so is cut more or less transversely. In this part there is some indication of the normal arrangement. In the centre of this part is seen the central artery and a very small vein surrounded by fibrous tissue (Pl. I, fig. 2). Here the nerve is intimately connected with its fibrous sheath above, from which it receives numerous intersections. Just in front of this region there are strands of fibrous tissue, passing downwards and backwards, which may represent a rudimentary *lamina cribrosa*, but this is situated much farther back than usual.

Between this and the disc the main mass of the nerve consists of a conglomerate mass of nerve-fibrils, nuclei and *corpora amylacea*.

The nerve fibrils are not separated by neuroglial septa, but run forwards mainly in the centre of the nerve ; they are most abundant in the median and temporal sections. Surrounding these fibres, and also to a certain extent scattered amongst them, are numerous nuclei of various shapes ; some form groups of small round nuclei, which simulate exactly retinal nuclei ; others are spindle shaped, whilst at the posterior part are areas close to the cross-cut nerve which are densely packed with nuclei of irregular shapes. These may be an irregular overgrowth of neuroglia. The whole

condition is probably due to proliferation of the cells of the inner layer of the optic stalk and the formation of very atypical retinal tissue.

On the temporal side of the nerve is a well-marked strand of fibrous tissue which is connected with the fibrous tissue entering through the gap in the pigment epithelium in the inferior temporal region, mentioned above. This strand runs forwards, gradually fading away at the disc, and is connected posteriorly with the fibrous tissue surrounding the A. centralis retinæ (Pl. II, fig. 4). This is probably the remains of the invaginated mesoderm in the optic stalk.

Scattered amongst these cells are a number of *corpora amylacea*, staining deeply with hæmatoxylin, varying in size up to 80 μ, for the most part circular, but some with pointed bosses (Pl. I, fig. 2). According to Parsons (7) such bodies are sometimes found in normal optic nerves, but are more common in atrophic ones. They are believed to arise from the neuroglial cells, and are also found in the central nervous system.

The retina.—The layers of the retina, instead of ending sharply at the edge of the disc, become irregular in arrangement and turn in along the nerve. Direct continuity can, in many places, be traced between the nuclei of the inner and outer nuclear layers and the groups of scattered nuclei in the nerve. Several folds of the nuclear layers are found close to the disc, and again on the disc itself; some of these form rosette-like structures, with an internal basement membrane corresponding to the external limiting membrane of the retina and an areolar structure in the centre which may represent the rod and cone layer (see Pl. I, figs. 1 and 2). Some of these in places resemble closely the rosettes occasionally seen in glioma retinæ.

The dural sheath of the nerve appears to be thicker than normal, and the position of the subdural space cannot be determined.

The arrangement of the *blood-vessels* in the nerve is as follows. In the posterior part of the nerve (the region cut transversely) there is a central artery accompanied by a small vein, surrounded by fibrous tissue (Pl. I, fig. 2, and Pl. II, fig. 3).

The central artery gives off a few branches, but no definite continuity between these and the vessels in the anterior part of the nerve can be traced as the series of sections is not complete.

In the anterior part of the nerve four arteries and two veins can be seen running forwards to the disc to supply the retina. In the superior part of the nerve are two arteries slightly to the nasal side, running forwards embedded in the atypical tissue which forms the nerve. Slightly to the temporal side of these and rather nearer to the edge of the nerve is a large vein which runs forward and divides into two in the retina at the edge of the disc. In the lower part of the nerve are also two arteries and a vein, this vein being situated at first centrally, but running on to the retina below and between the two arteries (Pl. II, fig. 3).

This arrangement of blood-vessels corresponds with that often found on the edge of the normal disc, namely, four branches of the A. centralis retinæ (two superior and two inferior), and two branches of the vein (situated to the temporal side of the arteries), upper and lower, the division of each of the two veins into temporal and nasal branches usually taking place beyond the disc. In this case the division of the arteries has occurred far back in the nerve (possibly in the region of the abortive lamina cribrosa mentioned above) instead of on the disc.

Further Discussion.

Although we are of the opinion that this case fits in with Coats's views on the possibility of abnormal differentiation of the optic cup and stalk, we have also considered the possibility of other explanations. Two only need be considered—(*a*) that it is a form of coloboma of the optic nerve, and (*b*) that it is due to a retraction of the retinal layers backwards through the papilla.

(A) *Coloboma of the optic nerve.*—A certain amount of confusion appears to exist in the literature as to the exact meaning of the term "coloboma." The term "typical coloboma" as applied to the posterior parts of the eye signifies a deficiency

apparently arising in connection with a delayed closure or non-closure of the fœtal cleft. A coloboma of the optic nerve should therefore, strictly speaking, arise from delayed closure or non-closure of that portion of the fœtal fissure which is normally included in the optic disc and nerve. In practice, however, other conditions are frequently confused with this. For example, a choroidal coloboma situated at the edge of the disc, a failure of mesoderm to enter the cleft in the optic stalk, or even an excessive pigmentation of the disc, might all be mistaken, ophthalmoscopically at least, for true coloboma of the disc, which appears to be a rare condition.

According to von Hippel (8), from a consideration of microscopically examined specimens, the ophthalmoscopic picture of coloboma of the nerve entrance is dependent on a cystic ectasia of the globe wall below a normal or slightly altered nerve, which enters from above nearly perpendicularly to the globe. The foramen scleræ is thus much enlarged. In the ectasia, choroid and retinal pigment epithelium are absent, but retinal elements may be recognisable. Nerve-fibres may pass over the ectasia to the retina below. In the nerve, the central vessels were missing five times out of the fourteen cases, coursing then below or in the sheath and piercing the floor of the coloboma. In other cases they entered the nerve abnormally close to the globe.

It is probable that most of von Hippel's cases were in reality examples of choroidal colobomata situated at the edge of the disc and causing secondary distortion of it, while those in which the central vessels ran below the nerve may have been due to failure of inclusion of mesoderm between the lips of the neural portion of the fissure, and not to its delayed closure. In the case under consideration in the present paper there is neither ectasia, failure of choroid, nor displacement of vessels, and it does not therefore fall into line with von Hippel's cases.

In describing three new cases microscopically as colobomata of the nerve, Coats (2) notes that the first two at any rate might appear ophthalmoscopically as "*crater-like holes at the disc.*" The abnormalities in these two cases consisted of

pockets in the nerve, placed in relation to the scleral margin of the scleral foramen, or when further back against the pial sheath: these were lined by pigment epithelium showing direct continuity with that in the globes, and were filled with a loose cellular tissue, suggesting retinal elements and neuroglia. The lamina cribrosa was defective over this area, which in sections involved in one case nearly half, in the other only a small fraction of the disc. In each case some isolated patches of pigmented epithelium and distorted retina were present close against the opposite wall of the nerve well behind the lamina cribrosa.

Almost all cases of "hole at the disc" (and a number have now been reported clinically in the literature—(9), (10), (11), (12), (13), (14), (15)) occur against the temporal margin of the papilla or just below it. In Coats's cases he described them as being below, but admitted that the orientation of the globes was impossible owing to the complete removal of the muscle tendons, and that he only placed them in that position because that seemed the most likely site.

Whether or not the position of a defect of the nerve head or of the choroid can be taken as a criterion of its connection with the fœtal fissure is doubtful. The optic vesicle is invaginated from below and slightly externally to form the optic cup. Later it is probable that the line of the fissure in its ocular portion becomes altered by unequal growth of the two sides of the retina, the temporal side increasing rather more rapidly, so that the fissure runs downwards and inwards. This has been seen by one of us to occur in birds (16). It appears to be very variable in man, and observers differ considerably concerning it. Many attempts have been made to show a rotation of the whole eye during development (Vossius, Strahl, Henckel), failure or excess of which would account for the varying positions of colobomatous defects. No definite conclusions have been arrived at, and it is probable that the condition is variable, since accounts of the position of entry of the art. cent. retinæ into the optic nerve behind the globe in the adult differ according to different observers, some stating that it is commonly below and to the temporal

side, others considering it to be immediately below the centre of the nerve. In the present case there appears to be some hint of a cleft in the lower temporal quadrant of the nerve, but no true coloboma.

Since Coats's paper several microscopical examinations of this anomaly have been described (Lauber (17), Szili (18), Seefelder (19)), and they show in the main the same characteristics as in his cases: in some there is an actual cavity communicating with the interior of the globe, varying in length and diameter: in others the cavity is filled by nerve-fibres or as in Coats's case by loose cellular tissue: some contain pigment, others do not. In all there is a defect of the lamina cribrosa in relation to the so-called hole. The vessels in the nerve are usually central.

Though some of the cases have been combined with colobomata in the other eye, and the majority of authors consider the condition as due to some abnormality in the neural cleft, it is difficult to see a relation between the two in all cases. Secondary holes or ectasia are common in the walls of the typical coloboma involving the papilla (20), but are probably merely due to greater bulging at the affected points.

Wessely (9) thought that "holes at the disc" might be due to a spacing round a cilio-retinal vessel, but it is unusual to find any vessel coming out of such a hole.

Szili (18) believed that the hole might be the remains of the cavity in the embryonic nerve-stalk.

A defect in the lamina cribrosa may account for some of the less marked cases, but this is perhaps more probably a consequence than a cause of the condition.

The case now reported is, we maintain, best explicable by Coats's theory: there is some evidence of a possible defect in the posterior part of the neural cleft, but this by itself cannot be held responsible for the extensive development of pigmented epithelium and retinal elements around the nerve itself, although it may be the result of a tendency to abnormal development: indeed, it would have been singular if there had not been some impairment in the closure of the cleft,

as the commencement of the abnormality must have dated from an early period of intra-uterine life.

(B) *Retraction of the retinal layers* through the scleral foramen from cicatrisation in the nerve or orbit would probably result in a stretching of that part of the retina which remains in the globe.

In the present instance the retina is well formed except for the ganglion cell and nerve-fibre layers; near the disc there are several clusters or rosettes suggesting bunching up of the retina rather than traction.

There are no adventitious adhesions attaching the sclera to orbital tissue, as one would expect to find if there had been an inflammatory mass involving the optic stalk.

The fibrous tissue near the cut end of the nerve on the temporal side is probably only an irregularity of the ensheathing mesoderm, but it is just possible it may have caused traction backwards as it has intimate connections with the nerve itself. The main mass of evidence is against such an explanation, though it is unfortunate that a greater length of nerve was not preserved.

Congenital pigmentation of the disc.—It is convenient here to consider shortly this subject, which has also been treated by Coats (21). From a review of the literature there are several varieties of this deformity.

(1) Isolated flecks on the disc are possibly due to pigmented lamina cribrosa, which is rare in man, but is common in the baboon and the pig (Ogawa (22)).

(2) Pigment in holes on the disc has been commonly noted, and, though in some cases the appearance may be due to shadow, in many it is due to actual pigment epithelium.

(3) A large dense patch of pigment continuous with the disc edge was described clinically in Coats's paper (21), and his surmise that it was due to a melanoma in connection with the choroid is borne out by the anatomical examination of a similar case by Oguchi (23).

(4) Pigmentation of the whole disc has been several times reported (quoted by Coats (22), see also Roll (24), Zentmayer (25)) but has apparently not been examined anatomically. We

suggest that the present case may give a clue to some of the members of this group, but it is unfortunate that the details of the ophthalmoscope appearance are not more definite. The dense lining of the nerve by pigment, in places several layers thick, the absence of a lamina cribrosa, the presence of presumably semi-transparent nerve-fibres in the centre of the nerve should certainly cause a very dark appearance to the nerve head ophthalmoscopically. Other cases of pigmentation of the whole disc may, however, be due to a pigmented lamina cribrosa.

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(3) Monthus and Opin.—*Arch. d'Opht.*, xxiii, 1903, p. 32.

(4) Seefelder.—*Ber. 37th Ophth. Gesell.*, 1911, p. 353.

(5) Buchanan.—*Trans. Ophth. Soc.*, vol. xxxvii, 1917, p. 166.

(6) Bowen and Carlyll.—*Brit. Med. Journ.*, June, 1910, vol. i, p. 1546.

(7) Parsons.—*Pathology of the Eye*, London, vol. ii, p. 659.

(8) *Graefe-Saemisch Handb.*, 2 Auflage, 1900, vol. ii, pt. i, Ch. IX, p. 27.

(9) Wessely.—*Arch. f. Augenh.*, lxv, 1910, p. 98.

(10) Gebb.—*Ibid.*, lxvii, 1910, p. 278.

(11) Williams.—*Ann. of Ophth.*, xxii, 1913, p. 638.

(12) Holloway.—*Ophth. Record*, vol. xxiv, 1915, p. 250.

(13) Claiborne.—*Ann. of Ophth.*, xxv, 1916, p. 299.

(14) Köhne.—*Zeitschr. f. Augen.*, xxxvi, 1916, p. 212.

(15) Harwood.—*Amer. Journ. of Ophth.*, vol. i, p. 572.

(16) Mann.—*Journ. of Anat.*, vol. lv, p. 115.

(17) Lauber.—*Zeitschr. f. Augen.*, xxi, 1909, p. 494.

(18) Szili.—*Ber. 39th Ophth. Gesell.*, p. 344.

(19) Seefelder.—*Arch. f. Ophth.*, xc, p. 129.

(20) Juler.—*Trans. Ophth. Soc.*, vol. xxxvi, 1916, p. 297.

(21) Coats.—*Roy. Lond. Ophth. Hosp. Rep.*, vol. xvii, p. 225.

(22) Ogawa.—*Arch. f. Augen.*, lii, 1905, p. 137.

(23) Oguchi.—*Ibid.*, lxiii, p. 160.

(24) Roll.—*Proc. Roy. Soc. Med.*, Ophth. Sec., November, 1916.

(25) Zentmayer.—*Amer. Journ. of Ophth.*, vol. ii, p. 201.

The President said this was a very unusual specimen, and the Society was glad to have the explanation from Miss Mann, who was devoting herself so particularly to the embryology of the eye. Until that was promulgated, he thought there must be some want of proportion between the enveloping mesoblast, which was developing the secondary optic vesicle or cup, and the cup itself, and, as it were, a portion of the secondary optic cup, towards the posterior pole, had been nipped, as a kind of knuckle, and hence the retinal tissues were developed in what, superficially and macroscopically, appeared to be the head of the optic nerve. He wondered whether there was any evidence of medullated sheaths along those nerve-fibres, or whether they were merely the axis cylinders, which one would expect to find anterior to the lamina cribrosa. That, however, was a question for pathologists and embryologists.

2. *Epithelial hyperplasia of the ciliary body.*

By Maurice Whiting.

(With Plate III.)

Cases of tumour of the ciliary body other than melanotic sarcoma are rare, and the question whether they are neoplasms or hyperplastic changes is of interest. In the case which is the subject of this note the tumour was discovered in the course of a routine pathological examination.

G. G—, a man, æt. 58 years, came to the hospital in September, 1919, with a corneal ulcer in the right eye. Later, in spite of treatment, a hypopyon appeared. The ulcer was cauterised twice, and the anterior chamber was tapped twice in addition

to ordinary local treatment. The condition gradually improved, and in December, 1919, he was discharged from hospital, the eye being still rather injected with a dense leucoma, the centre of which was vascular, shallow anterior chamber, tension normal, and vision only perception of light. On January 24th he returned with the eye painful and injected. On January 26th the eye was excised and fixed in formalin. It was subsequently imbedded in celloidin, and sections were stained in hæmatoxylin and eosin.

Macroscopic examination.—Cornea thick and opaque. Anterior synechiæ. Iris atrophic, with exudate on anterior surface. Early vitreous infection.

Microscopic examination.—Corneal epithelium very irregular and eroded in places.

The substantia propria contains many superficial and deep vessels, with much round-celled infiltration, especially at the limbus. Slight cellular exudate in the anterior chamber. The iris and ciliary body are also infiltrated, and there is some proliferation and migration of pigment. In the ciliary body on one side is seen a small oval cellular structure which is the subject of this note.

It is one millimetre long and slightly less in breadth, situated in the section entirely beneath the ciliary epithelium, from which it is separated by a thin layer of connective tissue containing dilated thin-walled vessels. It is partially surrounded by pigment, which is thickest on the external aspect.

The tumour itself is composed of oval and rounded spaces lined with flattened epithelial cells: these spaces are separated by a homogeneous substance which stains faintly with eosin and contains a few oval nuclei.

The question arises whether this is a genuine new growth or an inflammatory or degenerative hyperplasia, and, if the latter, whether it must be attributed to the recent inflammation or to some condition of longer standing.

Such tumours have been on several occasions discovered in the course of routine pathological examinations and have sometimes been described as adenomata or even epitheliomata.

Alt, in 1898, described a series of three cases, all of which

PLATE III.

Illustrates Mr. Maurice Whiting's paper on Epithelial Hyperplasia of the Ciliary Body (p. 101).

Fig. 1.—Low power. Ciliary body and iris.

Fig. 2.—High power. Tumour with pigment layer surrounding it.

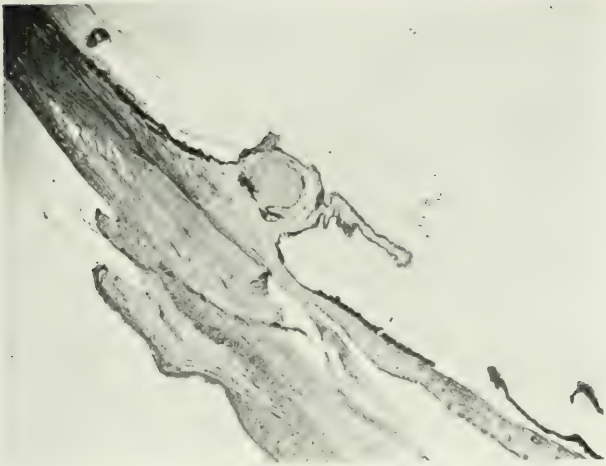

Fig. 1.

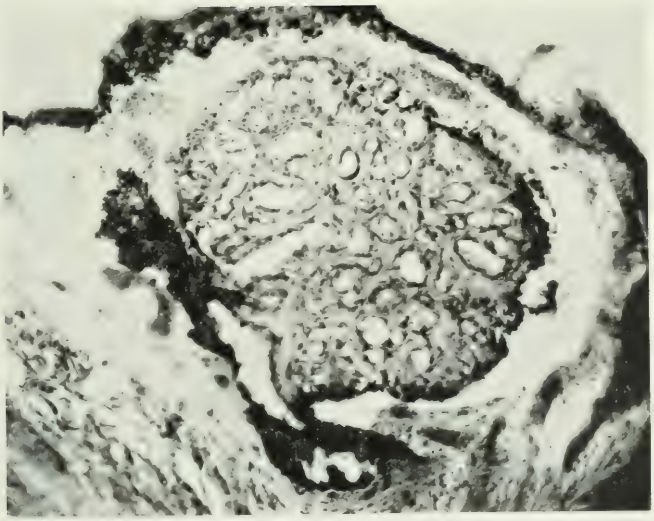

Fig. 2.

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were discovered accidentally during pathological examination, and had shown no clinical manifestations.

In the first case the patient, an old man, died of pneumonia, and a small tumour was found in the ciliary body of each eye.

In the second, also an old man, the eye was removed for sarcoma of the conjunctiva, while in the third the eye was removed as a result of panophthalmitis.

All four tumours resembled the one shown here, and Alt concluded that they were neoplasms, but not malignant.

Similar cases have been described by Parsons and Greeves, and the former in his work on the pathology of the eye concludes that such tumours are the result of inflammatory and degenerative changes, and should be classified as epithelial hyperplasias of the ciliary body. Other tumours of an epithelial nature have been described amongst others by Collins, Badal and Lagrange, Michel and Hine, but in all of these the nature of the microscopic appearances was essentially different from the present case, and in the last three the tumours were clinically evident.

It is often instructive to compare pathological changes in tissues in different parts of the body which have the same origin ; the choroid plexus in the ventricles and the ependyma of the ventricles may thus be compared with the neuro-epithelium covering the ciliary body.

Diffuse overgrowth of the stroma of the choroid plexus and of its lining epithelium with a flattening of the cells of the latter has been described by Delamarre as resulting from chronic inflammation. The ependymal epithelium in the ventricles may also show marked proliferation from acute or chronic inflammation.

In both the plexus and ependymal epithelium nutritional and inflammatory changes seem to pass by gradation into tumour processes. A large papillary tumour in the latter region was, however, considered by Brachanov to be a simple hypertrophy and hyperplasia, and tumours of any sort are rare. The same may probably be considered true of the ciliary body.

It is curious that while gliomas arising from the posterior

visual part of the epithelium of the optic cup are, though rare, a matter of constant clinical experience. neoplasms of the ciliary epithelium are so very seldom observed.

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Note.—The writer is indebted to Mr. Worth for permission to publish the details of this case. He had anticipated some discussion on the nature of this tumour from the pathologists among the members of the Ophthalmological Society. Failing this, it may be stated that the history of the case and the microscopical appearances of the section support the view that it is a hyperplasia of the unpigmented layer of epithelium covering the ciliary body. The mass grows into the ciliary body in much the same way that the epithelium does into the mesoblast to form the primitive lens. This explains the fact that the tumour is surrounded on only three sides by pigment.

3. *Some fundus drawings of papilladema.*

By Leslie Paton.

A complete series of coloured drawings, showing the development of papilloedema from its very earliest stages to its fully established condition, was shown by Mr. Paton by means of the epidiascope.

The PRESIDENT said it was well to be reminded of the detailed points in these cases in connection with papillœdema in all its stages, and to see depicted cases from their commencement or their early stage to the ultimate result. A point which members would be glad of enlightenment upon, if Mr. Paton could give it, was whether a decompression operation should, in the interests of vision, be recommended. When that problem came before him, he always found it difficult of solution. If one waited until vision began to deteriorate, it seemed that the subsidence of the papillœdema would be accelerated, but he thought the atrophy might be accelerated in the nerve-fibres. But as long as vision was well maintained, and the arteries were showing no signs of constriction, could one recommend an operation in anticipation that the vision might fail if there was any longer delay? And ophthalmologists were always faced by a considerable number of cases in which, in spite of intense œdema, the condition did subside, vision was unimpaired, and the case which was thought to be one of progressive cerebral tumour must pass into some different category, and no operation became necessary; moreover, any operation for decompression would have been undesirable.

One feature in papillœdema, which Mr. Paton did not indicate in his drawings nor allude to, was the exudation, which the speaker was accustomed to recognise along the perivascular lymphatic channels, and the organisation of that exudate into the well-known white lines along the attenuated arteries in the atrophic stage.

With regard to the question of the macular fan, he was under the impression that this was more frequent in papillœdema in young patients.

Mr. JOHNSON TAYLOR asked whether Mr. Paton, with his unrivalled experience of papillœdema, could diagnose, straight off, a case of pseudo-papillitis. All saw cases which looked exactly like optic neuritis. There were no hæmorrhages nor any exudation patches, but otherwise the appearance was typical of that seen in true optic neuritis. He had been taught by his revered teacher, Mr. Nettleship, that one could

only detect these cases satisfactorily by watching their course. He would be glad if Mr. Paton could say what was the exact pathological condition in pseudo-optic neuritis. Why was the disc in that condition so much swollen? Years ago he saw a case, diagnosed by a former President of the Society as optic neuritis, when it was only a very thin lamellar cataract.

Mr. Cyril Walker asked whether Mr. Paton saw cases in which papillœdema was associated with myopia—a matter which was conspicuously alluded to years ago by Dr. Rayner Batten. It had been a matter of surprise to the speaker, and he could not recall having seen such a case.

Mr. G. Maxted asked whether Mr. Paton could differentiate between papillœdema which was the result of intracranial pressure and a case of pure inflammatory optic neuritis. Frequently he was asked by colleagues in these cases which of these conditions it was due to, and he sometimes had difficulty in answering the question. Were there any clinical appearances which would help one to decide?

Mr. Leslie Paton, in reply, said he could not attempt a full reply to all the points raised, otherwise he would occupy the whole morning session.

In answer to the President, the settlement of whether the case should or should not be operated upon must ultimately rest with the physician, and it must depend on other signs than the papillœdema. The ophthalmic surgeon had to advise as to the prospects or as to the necessity of operation in order to save the patient's sight. There were one or two points of importance in regard to that. If the disc in a particular case had begun to show that opalescence which was associated with a slight narrowing of the calibre of the arteries, one could be certain that, whether operated upon or not, the disc would atrophy and blindness would result. But if the arteries were of good calibre, he believed the chance of saving the sight by operation was good. If there was stippling of the surface of the disc, without the opalescent whiteness he had referred to, the chances, again, might be fairly good. But when the last-named condition had set in, the prospect was bad.

There was another point of considerable importance in regard to the cases, namely, the prognosis in regard to life after operation, once the atrophy had become marked. To get a good result from operation in a case of cerebral tumour, it was very desirable to operate before failure of sight had commenced. If the atrophic stage had become marked, it seemed as if the contents of the cranial cavity had become congested, and if one suddenly relieved the pressure by operation, a bad vital result might ensue, *i. e.* a bad outlook as regards life.

He was glad to hear the President refer to the white lines. The appearance was common, and he regarded it as one of the most important signs of consecutive atrophy. In the last drawing shown, moreover, there were no traces whatever of perivascular infiltration in these vessels, there were no white lines, so these must not be regarded as an essential feature of consecutive atrophy. When they occur, the white lines are usually restricted to the vessels on the disc and the immediate neighbourhood, and there are no signs of them further out in the retina.

In answer to Mr. Johnson Taylor, as to the distinction between pseudo-papillœdema and true papillœdema, he wished he could make the distinction off-hand. On Monday he saw again a case he had seen twelve years before as one of cerebral tumour. On the former date he was in Queen Square Hospital for six months: he had violent headaches, and, as it was then called, optic neuritis, and during the six months and until now the disc appearances had not changed. The man had about 3 D. of astigmatism, for which the speaker gave him glasses. When seen a few days ago the edges of the disc were completely blurred all the way round, and he had to admit it was very suspicious of papillœdema. The man said his headaches were absent for five years. At about that date the man lost his glasses, and did not bother to have them replaced, and when he came last week he had a very violent headache. The disc appearances were absolutely the same as he had described them twelve years ago. He could not say even now that the man had not got true

papillœdema. At St. Mary's Hospital a case was admitted, under Dr. Wilfred Harris, and when the latter asked the speaker to see it, he said, "I don't know about this case; I have had it in two months, and if it had not been for the optic neuritis I should have regarded it as a functional case." He had a high degree of hypermetropia, and it was the most beautiful case of pseudo-papillœdema he, the speaker, had ever seen. Dr. Harris was quite right in his view; it was functional. Another case was seen at the National Hospital, and he asked some half-dozen colleagues to see it. None could be quite sure whether or not there was papillœdema. There was a little blurring of the discs, but very little swelling, and the appearance remained unchanged for two years. Ultimately the man died from a large cerebral tumour, so the condition in this case was a true papillœdema. Some showed definite differences between the upper and the lower edge of the disc and the extra-macular region. But in the normal disc there could be a difference of a dioptre between the upper edge of the disc and the extra-macular region.

He was somewhat at a loss to know how the question in regard to myopia had come to be believed, *i. e.* that myopic people did not have papillœdema. Possibly one explanation was that there might be much œdema in a myopic disc without much swelling. He had seen papillœdema of 5 D. and it had a good high myopic crescent. It commenced with hyperæmia of the central part of the disc, and in that case considerable œdema developed before there was any swelling. He was able to demonstrate to his class at Queen Square Hospital a case of myopia of 15 D. in one eye, 32 D. in the other, in which there was swelling of the disc. He, Mr. Paton, had entered into the question at some length in a paper. There was no reason why myopes should not have papillœdema; they got it quite as markedly as did other people.

Fig. 1.

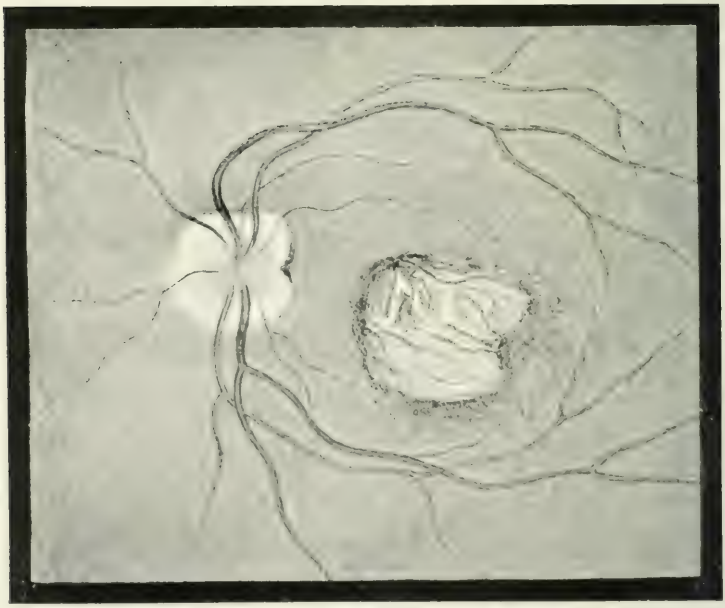

Fig. 2

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PLATE IV.

Illustrates Dr. Rayner D. Batten's paper on Symmetrical Destruction or Absence of Retina in Macular Region. ? Correlated to the Maculo-cerebral Degeneration of Adolescence and Infancy (p. 109).

4. *Symmetrical destruction or absence of retina in macular region. ? Correlated to the maculo-cerebral degeneration of adolescence and infancy.*

By Rayner D. Batten.

(With Plate IV.)

A. F—, æt. 67 years. V: R. $\frac{3}{60}$, < J. 12, not improved by lens. L. $\frac{3}{60}$, < J. 12, not improved by lens. Pupils equal, active; t.n.

In the two eyes there are symmetrical patches occupying nearly the whole of the macular area two to three times the size of the O.D. with apparently complete absence of retina. The margins of the area are clearly defined and dotted over with fine retinal pigment dots of a dark-brown colour (not black).

Thin choroid covers the floor of the area and numerous choroidal vessels cross it. There are no retinal vessels or other evidences of retina over the area.

The appearance suggests absence of retina over the area. The brown pigment spots I regard as typical of retinal as opposed to choroidal degeneration. Both O.Ds. are irregular in outline with a bulge towards the macula. This area is paler than the rest of the O.D.; ? atrophy of papillo-macular fibres.

History.—The patient is the fourteenth child in a family of sixteen; the others are believed to have good sight.

She is married. First child born dead; two miscarriages. Five children healthy to adult age.

Patient had severe epistaxis twenty-seven years ago, and again in March, 1921. Speech became affected October, 1921. Her vision has been poor for reading for the last five years. Memory poor.

She was referred to Dr. Feiling, who reports her symptoms suggest an early stage of bulbar paralysis or so-called pseudo-bulbar palsy from bilateral cerebral degeneration of muscular origin.

No absolutely definite signs in central nervous system found.

Re-examined in March, 1922, condition slightly worse. Very emotional. Extensor plantar response on the left side.

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5. *Congenital pigmentary plaques (moles) of retina (re-shown).*

By Rayner D. Batten.

Alice E—, æt. 40 years.

The case was originally shown at a meeting of this Society in 1893, and description and report of the case can be found in vol. xiv of the *Transactions*, together with a drawing by Mr. Holmes Spicer.

There is but little change in the condition of the plaques or fundus, but the central cups have enlarged and the œdema round the O.D. has disappeared.

Her refraction has changed from simple hypermetropia to one of hypermetropic astigmatism.

In 1893 R. + 2·5 sph. = $\frac{6}{6}$, L. + 2·5 sph. = $\frac{6}{6}$; in 1922 R. + 3 cyl. $\overset{165°}{\frown}$ = $\frac{6}{9}$, L. + 2.5 cyl. $\overset{150°}{\frown}$ = $\frac{6}{9}$.

She had what appeared to be rheumatic nodules on her head as a child. These appear now to have become ossified. She has had excision of the pelvic colon for carcinoma. She has had severe headaches all her life.

VI. INTRA-OCULAR TENSION.

1. *A suggestion for the management of the conjunctival flap in the trephining, etc., operations for glaucoma.*

By S. Johnson Taylor.

I have considerable diffidence in bringing this before the Society—in the first place on account of its simplicity, and in the next because, though unknown to me, as there is nothing new under the sun, it has very likely occurred to and been used by others.

Col. Elliot insists upon a large conjunctival flap in the operation he mainly devised, and let me for a moment here turn aside to say how deeply indebted to him and some others, *e.g.* Herbert and Lagrange, we are for these latter-day methods of dealing with chronic glaucoma. I lay emphasis upon *chronic* glaucoma, because, though I am well aware that the trephining, etc., operations are used by some surgeons in acute and subacute glaucoma, I still prefer and practise iridectomy in those conditions which, in the vast majority of cases done under deep local anæsthesia, as devised, I believe, by George Pooley and myself, is immediately and permanently satisfactory, and does not leave a fistulous communication with the interior of the globe which may in future years possibly lead to infection and trouble. I am well aware that there are some advantages in treating acute and subacute cases by trephining, etc. I will not enumerate nor mention them, as they are well known and sufficiently obvious, but the disadvantage I have mentioned outweighs in my opinion the advantages.

But that was a digression—now for the fact itself. We have dissected up our large conjunctival flap, we must take care

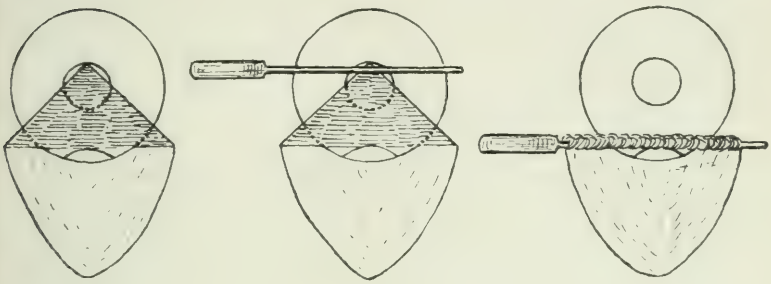
not to buttonhole or tear it away at its base—a risk we run if we draw it forwards at a right angle to the eye with a pair of forceps, however gently, and however smooth the instrument; if, on the other hand, we turn it down on the cornea and keep it fixed in that position by a spatula, etc., we cannot well see the pupil and iris at the upper part, nor readily note the change in shape and position of the pupil directly the trephine has opened the anterior chamber—a very valuable danger-signal.

It occurred to me last year that all these little difficulties and risks could be overcome if we could roll up the flap from apex to base on some sort of probe, and, making the attempt with an ordinary wooden match, whittled down to the size of a fine probe, I found it succeeded admirably, not only keeping the base of the flap well out of the way of the trephine and permitting a good view of any change in the shape and upward movement of the pupil when the anterior chamber is opened, but the base is so broad that the method ensures quite a strong and secure fixation of the eye without any risk of tearing. This will, perhaps, be especially appreciated in the performance of Ridley's "trench" operation, in which it is difficult to obtain satisfactory fixation of the globe with forceps during the scratching through of the sclero-cornea.

I have never used anything but an ordinary Bryant & May's wooden match, the head of which is decapitated and about half of the remainder whittled down to the diameter of a rather fine probe. The other half, square in shape, offers a good grasp to the finger and thumb of the left hand, and can easily be rotated thereby and so held. When operating on a right eye, I cut the handle part of the match a little shorter, as there is less room to work in on account of the nose looming largely, and I get the patient to look rather to the right. I practically do all my operations without assistance, but it would be quite easy, in the case of a right eye, to have the handle of the probe outwards and held by an assistant or by the operator's right hand. The match is boiled until it sinks, which renders it above suspicion from an aseptic point of view. I have used this method a good many times and

have had no trouble with it in any way; the flap rolls up quite easily to its base, and can be tightened or loosened as required by the least rotation of the handle part of the match. I recently did Ridley's trench operation with quite satisfactory results, but I am sure it would have been much more difficult to fix the globe in the lateral scratching through of the sclero-cornea if I had not used this rolled-up-flap method to steady the eye. As Mr. Ridley has quite candidly pointed out, his "trench" operation, though it does not leave a conjunctival bleb, with possibility of infection thereof later

FIG. 2.



1. 2. 3.

1. Conjunctival flap dissected up and turned down on the cornea.
2. Probe applied to apex of flap before rolling it up thereon.
3. Flap rolled up on wooden probe from apex to base, thus fixing it and using it as a means of fixation, whilst keeping its base out of harm's way and permitting a good view of the pupil, etc.

on, takes longer to perform than the trephining method; it certainly took me much longer, though I was not afraid to use a good deal of pressure when scratching through with the turned-up point of a sharp scalpel.

Mr. MALCOLM HEPBURN said the only point he would like to emphasise was the following. Anything which would ensure the stability and the firmness of the conjunctival flap was very much to be desired. The method described prevented all the tearing which was likely to ensue from taking hold of the flap by means of forceps. He had always felt that in

dealing with the flap the use of anything like toothed forceps was very dangerous, and there was therefore much to be commended in the method just shown by Mr. Johnson Taylor. He was now more convinced than ever that the important matter in trephining was to pay attention to the flap. If that were done carefully, so that there was no tearing or button-holing, then the results were far superior to those from any other operation.

The PRESIDENT said operators had felt anxiety about buttonholing the flap, or lacerating it by traction. He had tried to provide himself with a retractor for holding the flap downward and protecting its root from the anterior lip of the trephine: but it was difficult to do that with success. The instrument made was of a somewhat clumsier model than he had intended.

Mr. TRAQUAIR said that if the operator injected a few drops of novocaine solution beneath the flap which was to be made, the area became œdematous, so that one could grasp with forceps the swollen sub-conjunctival tissue at the top of the flap, and exert traction by a large number of connections without directly pulling on the conjunctival membrane itself. That largely obviated the danger of tearing.

Mr. GRIFFITHS agreed that Mr. Johnson Taylor's method got over a difficulty, but it could be got over better by turning the conjunctiva up from the cornea, instead of turning the flap down. One separated the conjunctiva from the cornea and allowed it to retract, leaving a clear field of view for the operation, afterwards fixing it down with a stitch. He discussed that method soon after the war ended, but he did not try it until two years ago. When in Italy he called upon Prof. Volero, of Florence, who told him he had used the method for several years, and showed him the technique. He had feared there might be leakage, but it did not occur. Perhaps members would like to try the method.

Mr. LESLIE PATON said he had used once only the procedure just described by Mr. Griffiths, in which he had button-holed

the flap. It was said that when button-holing had occurred, one should afterwards make the trephine hole away from the button-hole. He concluded that instead of trying a fresh trephine hole it was better to convert the case into von Mende's operation, cutting away down to the side, a little inside the button-hole, and then sewing the flap down. The case in which he did that turned out as well as any case after trephining.

Mr. Harrison Butler said that he held the flap with a pair of straight iris forceps without damaging it. After incising the conjunctiva he picked up the anterior fibres of the capsule of Tenon and held on to this structure with the forceps. They did no damage and were more convenient than other methods of retracting the flap, which in some cases were more liable to bruise the flap than the forceps. At one time he had difficulty in splitting the cornea, but he found that if the conjunctiva were gently stroked with a Landolt's scalpel, not in the exact line of cleavage—the crack he would call it—but just anterior to it, more towards the cornea, then the flap turned back with ease. It was only a question of about half a millimetre, which made all the difference. If one cut exactly in the crack, one seemed to come on to fibrous tissue and progress was slow. He had demonstrated this to Mr. Stack.

Mr. Stack: I can bear out all that Mr. Butler says.

Mr. Johnson Taylor, in reply, said he was satisfied with the method as he had devised it. It gave good fixation, protected the base of the flap, and gave a perfect view of the iris and pupil.

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2. *Some observations on the intra-ocular tension in cases of thrombosis of the retinal veins.*

By R. Foster Moore.

The following observations constitute a part of a general investigation of thrombosis of the retinal veins which was

commenced in 1913 under the ægis of the Lang Research Scholarship at Moorfields.

This investigation is now complete, and I hope to publish it in full at a later date.

Sixty-two patients were examined during that year, and, as far as possible, they have been kept under observation until now, or till the time of their death.

The present communication deals with only 37 of this number upon whom tonometric observations were carried out, and of them 18 were suffering from thrombosis of the central vein itself, and 19 were cases of tributary thrombosis.

The intra-ocular tension was taken with Schiotz's tonometer under cocaine anæsthesia. I had previously shown that the reading can be taken before the cocaine itself has any effect on the tension, and that in this respect holocaine has no advantage over cocaine (*Roy. Lond. Ophth. Hosp. Reports*, 1915, xx, 109).

It is clearly desirable that cases in which the central vein itself is thrombosed should be considered separately from those in which a tributary alone is involved, and so they have been arranged in separate tables; cases in which glaucoma ensued have been collected into a third table.

These tables show the tension of the affected eye in the first column, of the unaffected eye in the second column, and in the third is given the length of time from the onset of the thrombosis, and the intervals between the successive readings.

Thrombosis of the central vein.—Having regard to the rather frequent development of glaucoma in cases of thrombosis of the central vein, one was a little inclined to assume that the intra-ocular tension of those eyes in which thrombosis occurred, but which did not develop glaucoma, would be greater than that of the unaffected eyes, and it was therefore surprising to find that the tension was persistently less than the sound eye in the majority of cases. Thus, excluding those cases in which glaucoma ultimately developed, there were 12 cases on which periodical readings were made, and, taking them all together, the average tension for all the affected eyes was 17·25, and that of the unaffected eyes was 21·2, a difference

of 18 per cent., or, if the last case to which I shall refer later be omitted, the relative tensions are 15·7 in the affected eye and 21·3 in the unaffected, a difference of 35·6 per cent.

In the accompanying table the uniformity with which the difference between the two eyes was maintained over long periods will be seen (Table I).

This difference does not represent a very large reading on the Schiotz scale, and it has been shown by Priestley Smith that unless attention be paid to details of technique in the use of the tonometer, faulty readings are easily obtained, to the accuracy of which statement I can testify. Had these figures therefore been variable, or wanting in consistency, such as might possibly be attributed to faulty technique or personal error, one would have felt no confidence in attributing any particular importance to them.

It will be noticed that the readings were almost without exception not only less in the affected eye, but were approximately less by the same amount at the different times. There are two readings only where this is reversed. In the last case the tension of the affected eye was uniformly greater than the sound eye at each reading.

Having regard to the almost uniformly lowered tension in the case of central vein thrombosis, it is of interest to compare the tension of cases in which a tributary only of the vein became thrombosed.

Observations were carried out on 19 such patients, and the detailed results with the intervals between the readings are included in Table II.

An analysis of these patients shows how consistent are the readings obtained: all the readings, as in Table I, were made independently, and without reference to previous readings. I may here state my confidence in Schiotz's tonometer, and without prejudice as to the value of the readings as expressing actually mm. of Hg., a point which Priestley Smith has worked out with characteristic skill and care, I am confident that for relative readings the instrument is one of great accuracy and delicacy.

The readings show that with one or two individual excep-

TABLE I.—*Cases of Thrombosis of the Central Retinal Vein, to show the Intra-ocular Tension in the Affected and in the Unaffected Eye.*

The figures are in mm. of Hg. according to the Schiotz scale.
In the last column is given the interval between the onset of symptoms and the first reading and the intervals between the successive readings.

Name.	Affected eye.	Unaffected eye.	Interval between the readings.
Charles Foulger	16	22	4 months.
	15	22	3 weeks.
	15	24	5 „
	15·5	19	3 months.
	18	31	2 „
	13	23	7 weeks.
	13	18	17 months.
Jesse Gould	17	17·5	6 weeks.
	14	16	1 month.
	17	19	2 months.
	17	18	2 „
Seely Davenport	17	32	6 months.
	17	32	4 weeks.
	16	27	8 „
Elizabeth Holmes	17	22	2 weeks.
	17·5	25	3 „
	17	17	3½ months.
Isabel Merriman	12	15	—
Betsy Reuben	17·5	17·5	5 months.
	12	14	14 weeks.
	14	14	19 months.
Jane Couch	17	19	2 months.
	15	19	2 „
	15	21	2 „
Elizabeth Carter	15	—	1 month.
John Hancock	10	13	2 months.
	11	15	1 month.
Robert Cole	13	12	9 years.
Francis Malivari	22	22	Some years.
Albert Young	21	36	2 months.
	17	28	1 month.
	15	32	2 months.
Annie Berry	25	18	6 months.
	27	22	2 „
	33	25	3 „
	22	19	5 „
	23	18	7 „
	25	18	4 „

tions there is no essential difference in the tension of the two eyes in this group of cases (Table II).

If one takes an average of all the readings for the two eyes, the tension of the affected eye is 15·3, and that of the ununaffected eye 15·0, *i. e.* the affected eye has a tension of, roughly, 2 per cent. greater than the unaffected.

This is perhaps a rather gross way of looking at the matter were it not for the consistency of the readings in the individual cases.

It becomes clear, then, that in cases of thrombosis of the central retinal vein the tension of the affected eye is 18 per cent. less than in the sound eye (or if the last case be excluded it is 35·6 per cent. less), but in cases in which a tributary only is involved there is no essential difference beween the two eyes: the affected eye, in fact, has a tension which is 2 per cent. greater than the sound eye.

It is well known that a characteristic form of acute glaucoma follows thrombosis of the retinal veins in a certain proportion of cases.

Of the present series there were 18 cases of central vein thrombosis upon whom tonometric observations were carried out, and of them 5 subsequently developed glaucoma (Table III). These have been collected into a separate table. In no case did glaucoma follow thrombosis of a tributary.

One wondered whether it would be found that those eyes which ultimately became the subject of glaucoma would perhaps from the first show a tension which was greater than the sound eye, or, failing this, whether the tonometer would show that the tension of the affected eye increased beyond that of the sound eye, before there were any subjective symptoms or other clinical signs of the onset of glaucoma.

On looking through these cases it will be seen that in Case 5 the other eye was previously the subject of glaucoma, and in Case 3 there was a single reading only, which showed, however, that the affected eye was lower in tension than the sound eye at this date.

In Case 2, however, three weeks after onset, the affected eye was equal in tension to the sound eye, instead of being

TABLE II.—*Cases of Thrombosis of a Tributary of the Retinal Vein, to show the Intra-ocular Tension in the Affected and in the Unaffected Eye.*

The figures are in mm. of Hg. according to the Schiotz scale.
In the last column is given the interval between the onset of symptoms and the first reading and the intervals between the successive readings.

Name.	Affected eye.	Unaffected eye.	Interval between the readings.
William Garrett . .	19	19	12 months.
	22	22	1 month.
	21	23	2 months.
	17	13	2 ,,
	16	17	8 years.
Alfred Keeling . .	13	13	2 weeks.
	18	18	4 months.
	14	12	3 ,,
	12·5	14	18 ,,
— Albinson . . .	13	13	7 years.
Louisa Field . . .	14	17	16 months.
	17·5	17·5	1 week.
	15	13	4 weeks.
	18	18	8 ,,
Elizabeth — . . .	13	14	3 months.
Charlotte Wall . .	13	15	1 month.
	15	18	6 weeks.
	15	21	4 ,,
Sarah Harris . . .	16	16	3 weeks.
	15	15	4 ,,
Emma Rayment . .	13	13	7 months.
	18	18	6 weeks.
	12	11	8 months.
William Foxlow . .	13	13	17 years.
Ellen Warner . .	15	15	—
Charles Swan . . .	13	15	1 week.
	12	15	4 weeks.
	13	15	1 week.
	14	17	1 ,,
	12	15	2 weeks.
	12	13	15 months.
Alice Heffer . . .	19	19	6 ,,
Richard Stephings . .	7·5	12	—
Charles Sothern . .	13	11	12 months.
Frances Morris . .	15	15	1 month.
	13	13	1 ,,
Caroline Mansfield .	15	15	3 months.
	15	15·5	3 ,,
	14·5	14·5	3 ,,
	15	15	4 ,,
Mary Reynolds .	8	6·5	5 weeks.
	12	8	2 months.
	11	7	2 ,,
	12	12	7 years.
Louis Woolfsohn . .	25	15	4 years.
William Waghorn . .	30	22	19 months.
	32	18	1 month.

TABLE III.—*Cases of Thrombosis of the Central Retinal Vein in which Glaucoma ensued.*

Name.	Affected eye.	Unaffected eye.	Interval between the readings.
Elizabeth Valentine .	16	22	3 weeks.
	15	22·5	1 month.
	45	17	3 weeks.
	37	15	1 week.
	38	—	1 ,,
	55	—	2 weeks.
	66	—	2 ,,
	59	—	3 ,,
	60	17	3 ,,
James Popkin . .	24	24	3 weeks.
	33	25	2 ,,
George Neate . .	22	31	5 weeks.
Thomas Grainger . .	20	17	9 weeks.
	22	17	2 months.
	22	13	1 month.
	55	12	11 weeks.
	55	15	3 weeks.
	47	15	4 ,,
John Potter . . .	52	22	—
	52	40	—
	55	22	11 months.
	55	32	10 ,,
	140	100	5 years.

less than it as is usual, and two weeks later it was markedly greater than the sound eye, though there was as yet no other indication of glaucoma.

In Case 1 it will be seen that in the earliest stage the affected eye was softer than the sound eye, but that its tension was increased before there were any other indications of the onset of glaucoma; and in Case 4 the tension was increased above the sound eye from the first.

It is obvious that these cases are insufficient in number to formulate any dogmatic statement upon them, but, having regard to the uniformity with which the intra-ocular tension is reduced in central vein thrombosis, I think it may be said that in cases of this occurrence the presence of an intra-ocular

tension which is equal to or greater than the sound eye at any stage should lead one to fear the onset of glaucoma.

This appears to me to be a point which is worth further inquiry, so that a definite conclusion with regard to it may be arrived at.

The last case in Table I needs special mention in this connection, for, as it will be seen, the tension of the affected eye was persistently greater than in the sound eye for 27 months for certain ; nevertheless, there were no signs of glaucoma at the end of eight years.

Relation of the intra-ocular tension to the general systolic blood-pressure. In a previous paper (*Roy. Lond. Ophth. Hosp. Reports*, 1915, xx, 115) I have shown reason to believe that the pressure in vessels of the size of the retinal and choroidal arteries does not vary, as between individuals, with the general blood-pressure, but that indeed with a high pressure in the brachial artery, the pressure in vessels of the size of the retinal arteries is often—perhaps always—less than the normal, and at the same time I showed that the intra-ocular tension did not vary with the general blood-pressure—that indeed a high or very high systemic blood-pressure was not associated with an intra-ocular pressure which was at all raised.

For this last purpose a group of 97 patients were first divided into those with a blood-pressure which was less than 180 mm. and those whose pressure was greater than 180 mm., and the intra-ocular tension of the two groups was compared.

Afterwards, using the same 97 patients, they were divided into those whose pressure was less than 160 mm. and those whose pressure was greater than 220 mm., those with pressures between these figures now being omitted from consideration.

A precisely similar investigation was carried out on the present smaller groups of patients, 32 in number.

The former table and the present one are put together in Table IV. Of the present cases the intra-ocular tension of the sound eye only was taken.

It will be seen that in the previous investigation the intra-ocular tension of those with a blood-pressure of greater than

TABLE IV.—*A Table to show the Relation between the General Systolic Blood-Pressure and the Intra-ocular Tension.*

The table comprises thirty-four patients in whom the necessary observations were available.

The blood-pressure and the intra-ocular tension represent in most cases the average of a number of readings.

In (i) the patients are divided into two groups—those whose blood-pressure was greater than 180 and those in whom it was less than 180.

In (ii), in order to draw a sharper distinction, the same patients are divided into those whose blood-pressure was greater than 220 and those in whom it was less than 160, those with intermediate pressures being excluded.

	Number of patients.	Average blood-pressure.	Average intra-ocular tension.	Difference in intra-ocular tension.
(i) Blood-pressure < 180 mm.	18	157	19	—
Blood-pressure > 180 mm.	14	220	18	Less by 1 mm.
(ii) Blood-pressure < 160 mm.	11	148	18	—
Blood-pressure > 220 mm.	7	244	16	Less by 2 mm.

A similar table to the above published in the *Royal London Ophthalmic Hospital Reports*, 1915, xx, i, p. 115:

(i) Blood-pressure < 180 mm.	48	143	14·8	—
Blood-pressure > 180 mm.	49	221	16·1	Greater by 1·3 mm.
(ii) Blood-pressure < 160 mm.	37	138	14·3	—
Blood-pressure > 220 mm.	26	244	15·3	Greater by 1 mm.

220 mm. was 1 mm. greater than those whose blood-pressure was less than 160 mm. In the present smaller group the intra-ocular tension of those with a blood-pressure of greater than 220 mm. was 2 mm. less than those with a blood-pressure which was less than 160 mm.

It may, I think, be confidently asserted that whilst no doubt the intra-ocular tension varies directly with the pressure in the intra-ocular vessels, as has been shown experimentally by Parsons and others, and as can be shown clinically by pressure on the carotid artery in man, a general systolic pressure, however high it may be, produces no increase in the intra-ocular tension.

Conclusions.—1. In most cases of thrombosis of the central retinal vein the intra-ocular tension of the affected eye is appreciably less than that of the unaffected eye.

2. This difference in the tension is maintained for long periods.

3. There is some evidence to show that in those cases in which acute glaucoma subsequently develops the intra-ocular tension of the affected eye, instead of being less than in the unaffected eye, is either equal to or greater than it from an early stage, and at a time when there are no other signs or symptoms which suggest that glaucoma will ensue.

4. In cases where a tributary only of the vein is thrombosed, there is no sensible alteration in the tension of the affected eye as compared with the sound eye.

5. These cases support previous investigation, which shows that a high systemic systolic blood-pressure is not in any way associated with an increase in the intra-ocular tension.

The PRESIDENT said these were conclusions on a subject which Mr. Foster Moore had made specially his own. Probably no one present would be able to controvert his views, but there might be some who could supplement them. The author had not said why there should be the secondary glaucoma of which he spoke. Perhaps, however, that did not belong to the present discussion.

Mr. BERNARD CRIDLAND, referring to Mr. Foster Moore's statement as to the relationship between the systemic blood-pressure and the intra-ocular tension, said he would like to mention that he had notes of a few cases which had been under observation for a number of years; they had troubled him very much at first, because he thought they were very suspicious of early chronic glaucoma. He had referred to these in a short paper he published some years ago in connection with the Schiotz tonometer. In the cases the tension had varied between 25 and 30, but there had been no reduction in vision, though the patients were examined on a number of occasions; and there were no other signs of chronic glaucoma; but, as the years went on, the tension became normal—below 25—though the patients developed general high blood-pressure. The three or four cases now referred to represented the other

side of the question. They had no increase of blood-pressure at the time he first saw them.

Mr. JOHNSON TAYLOR asked if Mr. Foster Moore would say why in cases of thrombosis of the central vein the intra-ocular pressure was generally lower. He would have thought it would have been higher.

Mr. BEATSON HIRD desired to thank Mr. Foster Moore for this very interesting contribution. The author did not say anything with regard to treatment, though that might be outside its scope. In his own experience he had seen four or five of these cases of glaucoma following thrombosis of the central retinal vein; and they got well, the tension coming down, even below the normal, without operative interference. He regarded such interference as serious and to be avoided. He would like to ask whether that was the general impression, *i. e.* that it was better to treat these acute glaucomas by local remedies, without operation. As a rule these patients were in acute pain.

The PRESIDENT said his experience would be that the operation generally called for in late stages was excision. He agreed that a palliative operation might be attended with success, but scarcely so in a blind eye.

Dr. BURDON-COOPER said he had done some experiments on these physical constants of the aqueous in glaucoma generally and in cases associated with hæmorrhage, and he put forth as of some interest the fact that the surface tension of the aqueous humour was much reduced in glaucoma, and in the cases referred to it was below the normal. Assuming the surface tension of water to be 70 dynes per lineal centimetre, he found the surface tension in glaucoma to be about 62. And if the albumen in a fluid state was instrumental in hindering osmosis, he thought the old ideas held as to that must be given up. Perhaps these remarks might be of help to Mr. Foster Moore in his work.

Mr. CLAUD WORTH said he had recently had a case of glaucoma following thrombosis of the central vein. The only question seemed to be the relief of the pain. The depth of the anterior chamber appeared to be normal. As a palliative

measure he tapped the anterior chamber and used hot bathing. This caused the pain to subside, and it did not return.

Mr. Foster Moore, in reply, said Mr. Johnson Taylor appeared surprised that the tension in eyes in which thrombosis of the vein occurred was less than in the fellow eye: but that was what he, the speaker, would have expected. With regard to treatment, he did include a reference to that in his paper, but on account of the value of time he did not read it. As to operative treatment for these cases, he suggested whether, when the intra-ocular tension in an eye with thrombosis of the central vein was greater than in the other eye, it might not be advantageous to trephine at that stage. It might not be, but he did not think there was any treatment but that short of removing the eye.

VII. REFRACTION.

1. *Refraction without cycloplegics.*

By T. Harrison Butler.

Many of the subjects which we discuss at these Congresses, though of deep scientific interest, are somewhat academical, and neither vitally concern the well-being of our patients nor touch our reputation with the public.

The matter treated in this paper is one which may not only affect the comfort and well-being of those who consult us, but may eventually exert an influence upon our professional success. Under the circumstances it behoves us to adopt reliable methods—methods which give accurate results with the minimum of expense and discomfort to our patients, and do not demand an inordinate amount of time.

The main object of refraction is to give our clients the sharpest acuity, and above all to make them comfortable. I wish to emphasise the fact that we are not trying to solve a purely optical problem, but are trying to correct a defect which is not wholly dependent upon the dioptric system of the eye. There is also a psychic element, and its treatment demands a judgment which develops with practice. From this it follows that a technique suitable for a surgeon of ripe experience may not be safe or desirable for the beginner, however able he may be.

My object in writing this paper is not to advance any new facts or theories, but to promote a discussion upon a vital question: *To what extent shall we employ cycloplegics in refraction?*

In this connection we may divide ophthalmologists into three groups: Those who use a cycloplegic in the majority

of cases; those who use them frequently; and finally, those who avoid them in the large majority of cases.

As far as I have been able to judge by a study of the work of many of my colleagues, the results obtained by the three classes are exactly the same. I have seen a large number of refractions carried out by two surgeons of high eminence who rarely use a cycloplegic, and I have found their work reliable. I have also seen many patients whose glasses have been ordered by an apostle of homatropine, and here again I find the same degree of accuracy.

Judged, then, by the standard of actual practice, there is no call for the routine use of a cycloplegic. My personal work leads me to a similar conclusion.

Ten years ago I was using atropine and homatropine for well over half my cases. Gradually I have used them less and less, till to-day I hardly call upon them once a month for adults, and not often for children over ten, and even younger, if they are intelligent. I have been able to review many of these cases after years, and, as far as I can judge, the results with a cycloplegic are not one whit better than those without it. I have worked out hundreds of cases with and without homatropine, and in but a few has the drug helped me. In many cases I have got less hypermetropia after the instillation of a 2 per cent. solution of homatropine three times in an hour than I did without it.

I have naturally detected occasional errors in my work without a cycloplegic, but I have found at least as many, if not more, after the use of homatropine.

I was encouraged to give up cycloplegics by my friend, the late Mr. Devereux Marshall, who told me that he hardly ever used them, and regarded them as unnecessary in the majority of cases.

Why should we use cycloplegics? We have all been brought up under the shadow of the bogey *spasm of accommodation*. Hirschberg tells us that this condition is one of the rarest in ophthalmology. I can recollect only two or three cases of real spasm, and both were in highly neurotic women. In the first I was not able fully to control it with atropine,

and the second woman was most uncomfortable with her hypermetropia under-corrected to the extent of 1 D. It was some weeks before she was able to wear her glasses with success.

I think that few will deny that real spasm of accommodation is so rare and so very obvious when present that it can be entirely disregarded as a controlling factor in our choice of method. On the other hand, there most certainly is such a thing as *a habit of accommodation*, an *excitable accommodation*, and perhaps even an *irritable accommodation*.

A child with, say, 8 D. of hypermetropia has acquired what we may call a *habit of accommodation*, and if we put up, say, lenses of 6 D., he will certainly refuse them, and may only with difficulty be persuaded to take 2 D. Fortunately in such a case a properly conducted retinoscopy will furnish practically the full amount of hypermetropia without the use of a mydriatic. I shall revert to this later on.

A second reason advanced for the routine use of a cycloplegic is that small degrees of astigmatism can be and are overlooked, whereas they become manifest after the use of homatropine. I can only say that this is contrary to my experience, and even appears to violate the principles of optics.

In all optical instruments, cameras, microscopes, and such like, a diaphragm gives more precise definition. A dilated pupil may blur definition, and some patients will read a line more with a normal pupil than with a fully dilated one. For this reason a small change of cylinder should be more easily appreciated by a patient with a mobile than with a paralysed pupil. This is more evident if the test-types are brightly illuminated, for in this case not only is the image less defined with a dilated pupil, but there is apt to be some degree of dazzle.

The amount of confusion caused by a dilated pupil when there is an error of refraction is extraordinary. Quite recently I had a drop of cocaine instilled into one eye for the removal of a foreign body from my cornea. With a myopia of 2·5 dioptres I am perfectly happy without my glasses—in fact, I generally do not use them; this in spite of the fact that

my uncorrected acuity is $\frac{6}{36}$. With a pupil dilated with cocaine I felt quite blind in one eye, and the acuity was under $\frac{6}{60}$. With my glasses I got my usual acuity of $\frac{6}{3}$ partly.

As regards retinoscopy, I am quite certain that I can be more accurate with a moderate-sized pupil than with a dilated one. The shadows are sharper, and there is no confusion between the central and peripheral shadows. Scissor movement is rare, and, when present, is more easily interpreted when the pupil is small. The sole advantage of the dilated pupil is that the shadow is longer, and in consequence it is easier to judge the angle. It is true that in some cases—a very few—the pupil is too small for retinoscopy. This is found in some aged persons, and here a drop of cocaine may solve the difficulty.

The disadvantages of cycloplegics are sufficiently obvious. From the surgeon's point of view there is a serious waste of time, and a day's appointments may become muddled if several patients are waiting with drops and there is no vacant time for them. Most patients object to drops and often ask not to have them used. In some cases they are seriously inconvenienced: busy men and women naturally object to the loss of a working day, and even more to being deprived of an afternoon's golf or tennis. Occasionally the effect of homatropine persists for two or even three days, and may cause great annoyance—perhaps even the loss of a county cricket match!

Many children are frightened by the instillation of drops. They may in consequence become difficult to manage and the cycloplegic defeats its own end.

There is one vital objection to a cycloplegic. One has to guess how much to allow for the drug in ordering the glasses. In fact, a post cycloplegic test is necessary. Without a subsequent test the glasses must be a guess. In children I allow 1 D. for atropine: in adults less: but in cases of low myopia this correction may be wrong. In some cases patients require the full amount found under atropine, but others will submit to a reduction up to 1 D. In other words, we must either work out our cases fully before using

the cycloplegic or we must see them when the effects have passed off. In hospital work this entails double work for each patient, and in private practice it has even more cogent disadvantages. A busy man cannot afford to give a second consultation without a second fee, and these fees are not always possible, and are daily becoming more impossible!

Finally, there is in patients over forty-five a real danger of inducing glaucoma. I have had three cases in my hospital practice. In one case atropine was used in a case of corneal ulcer and glaucoma developed; in another it was used to dilate the pupil in a diabetic for fundus examination, and in a third my assistant instilled homatropine for refraction. Acute glaucoma developed in both eyes; iridectomy was done at once; but one eye was lost. The subsequent use of a meiotic has disadvantages of its own. The struggle between mydriatic and meiotic may be most uncomfortable, and may cause fainting, nausea, and vertigo. I have three definite examples in my mind, and in one case it happened twice. The patient went home, and was so upset that he had to go to bed. It would appear, then, that there is no real advantage in the routine use of cycloplegics, and many disadvantages.

I am now following my views to the logical conclusion, and in two of my hospitals I now rarely use a cycloplegic. I have just had a new eye-room at the Warneford Hospital, and the dark room is 25 ft. long. For the past six months I have worked without homatropine; except for children I have not employed it a dozen times. As far as I can judge, I am getting exactly the same, if not better, results than I did with homatropine, and the work is done in half the time.

I shall now outline my methods and then discuss the conditions that demand a cycloplegic.

Refraction with and without cycloplegics in my opinion depends upon accurate retinoscopy—an accuracy which, given good eyesight, comes with practice if adequate methods are chosen. If we are working without a cycloplegic, we must adopt measures which give complete relaxation of accommodation.

The first essential is a long room—the longer the better.

The orthodox " dark-room " is useless. The patient is seated in a dark corner, but the room itself must be dimly lighted so that objects upon the wall opposite can be seen with some distinctness. I use a red and a white disc about 4 in. in diameter—one for the right, the other for the left eye. For children pictures are useful.

The light must be small, and sufficiently bright to compensate for the loss of size. The conventional frosted bulb is bad, and does not allow of extreme accuracy. I use a focus-lamp in a Thorington's chimney fitted with an iris diaphragm. When this chimney is used, there is not enough light for writing and to see the lenses, so a shaded reading lamp is a convenience.

The surgeon must be able to use either eye with equal facility. It is absolutely impossible to perform a retinoscopy near the macula of the left eye if the surgeon uses his right eye, and unless the refraction is made at the macula it may be very inaccurate. I wish to emphasise this factor, for many surgeons confine themselves to the right eye. I for many years used the right entirely, and was always inaccurate with the left retinoscopy. I determined to use the left as well. It was not easy, but now I can use one as well as the other.

I think that a large flat mirror with a very small hole is the best. I have tried various types—plain-flat, amber-flat, cylindrical, and Lister's slight-concave. I believe that some still use the full concave mirror, but I am unable to get any good results with it.

I work at arm's length for two reasons. It is most convenient, especially if one has a lot of school clinic work to do, and it is a constant length. I deduct 1 D. from the turning lens, and I find that generally this gives the true refraction. In some cases this deduction is not sufficient.

Retinoscopy has its limitations. Under favourable circumstances it is possible to work within a limit of $\frac{1}{4}$ dioptre, but $\frac{1}{4}$ is the average. In some eyes, especially low degrees of myopia, the possible error is greater, and in some cases the result is wholly inaccurate. I have one case in mind in which, both with and without atropine, retinoscopy gave

a myopia of 3 D., but the patient, a lady of about 30 years of age, was emmetropic and had an acuity of $\frac{6}{3}$ partly. I can only imagine that in this case the macula was perched upon a small eminence. In some cases, although retinoscopy gives astigmatism with the rule, the patient will not take a cylinder. In one case I got my assistant to work the refraction out independently, and he got an identical result.

The chief, and perhaps the only common source of error in retinoscopy, is failure to refract upon the macula. This will in some cases introduce a large error, and this is especially true in high myopia.

Indications for the use of a cycloplegic.—When the retinoscopy is not absolutely positive, and when the turning glass is not obtained at once, but only after some coaxing, or when it varies, then I consider that I am not getting full relaxation and use a cycloplegic.

When the objective and subjective examinations do not agree within safe limits, then I return to the retinoscopy, and, if I still cannot get adequate correspondence, then again a drug must be used.

The use of the wing-test should afford some indication of a tendency towards spasm, but I am not at all sure that it does. One would expect excess of accommodation to be accompanied by excess of convergence. I make a practice of using a near test for heterophoria in every case, and when I get esophoria I am suspicious that there may be some concealed hypermetropia. When I get a little exophoria I feel safer.

A definitely neurotic type of patient is one likely to suffer from an irritable accommodation, and this in my experience is far more frequent at about the onset of presbyopia than earlier in life. In some of these cases homatropine is necessary, and in the younger type I do not hesitate to use atropine.

If I am compelled to use a cycloplegic, I prefer to choose atropine, unless it is contra-indicated by social or other reasons.

I regard homatropine with considerable suspicion. I think that in many cases it certainly stabilises the ciliary muscle,

but not in the position of full relaxation. I have a large number of records amounting to hundreds in which I have obtained less hypermetropia after the instillation of homatropine than I did before. I use a 2 per cent. solution, and instil it every quarter of an hour for an hour or more. I do not use oily solutions, because I dislike messy things and because the oil complicates the optical examinations.

I regard it as a useful but uncertain friend. I am not at all convinced that in some cases homatropine may not induce some spurious astigmatism, and, far from enabling us to detect low-grade astigmatism, it may produce it. I am not prepared to state that this is a fact, but I have long suspected it, and I did so at a time when I was employing it in most of my cases.

Children are far more intelligent than many imagine, and, being free from preconceived ideas, are admirable subjects for refraction. With the exception of high-grade hypermetropes, I believe them to be free from anomalies of accommodation. I am quite convinced that if a child has sufficient intelligence and concentration to look at a distant object, he is relaxing his accommodation. Under these circumstances I rarely use atropine and never homatropine. There is no fixed age below which a cycloplegic is necessary, but on the average in private practice I place it at eight. I had no difficulty in working out the refraction of one of my daughters, æt. $4\frac{1}{2}$ years. One eye had an astigmatism of $\frac{1}{4}$ D., the other of 7 D. The child was fully able to choose the best glass for the eye with the least astigmatism, and to state with absolute precision the angle of the large cylinder. I confirmed this result with atropine and found that I had received no assistance in ordering the correction. It is impossible to refract children in hospital without atropine, because there is not the absolute quiet that is necessary, nor is there time for the work.

As regards *myopia in children*. Here we have the class of case in which according to the books atropine is absolutely necessary and where most mistakes are made. I wonder why. A myopic child is not liable to spasm, and could not in the nature of things be expected to have any. Here we are on

safe ground, and we can fearlessly follow our retinoscopy, backed up by the result of the test with the glasses. We order the glass which gives the best acuity. It is of great importance to order an accurate spherical correction in myopia because under-correction means poor acuity. We can only do this by an objective test. It does not matter one whit if we reasonably under-correct hypermetropia in a child. If we correct most of it we have relieved all eye-strain, for the child has an immense reserve of muscle power to correct the remainder; but if we do the same with myopia we have failed in our treatment, and an intelligent child will soon tell us so!

The average boy or girl who goes to a preparatory school is the type I have in mind. I rarely use a cycloplegic for them, and they give me no trouble. I have seen many of them again after years, and I have so far discovered no material errors in the work. In all such cases I make certain that there is a reasonable correspondence between retinoscopy and subjective testing. I am not suggesting that a child should be allowed to choose his glasses unaided by science.

With regard to the use of the ophthalmometer, I have nothing against it, and, never having used it, I am not competent to express an opinion upon it. But I would like to ask one question from its advocates: Why, if it is accurate to within $\frac{1}{2}$ D., as we are told it is when correctly calibrated and used—why should it be necessary to use a cycloplegic to bring out the last vestige of astigmatism? No cycloplegic can affect the cornea, and the instrument only measures the corneal curvatures. I can only assume that the advocates of the machine do not trust it, but realise that there is such a thing as lental astigmatism. This is the variable factor which vitiates the results of the ophthalmometer, and can only be eliminated by the patient at the test-types. Why, then, spend £40 on an instrument when for a shilling you can buy a small mirror, cut a hole in the paper back, scratch off the silvering in the centre, and possess an instrument which will in capable hands give you the total error, both spherical

and cylindrical? I have such a mirror which I made in an emergency, and it is the best I possess.

In conclusion, I suggest that refraction without a cycloplegic is as accurate as that with one if only correct methods are employed, and if sufficient experience and skill are at the command of the refractionist. If not, then many of the most eminent men in this country and in others must be making countless mistakes, and some of us should have the opportunity of correcting them. I confess that they have not come my way.

I think that the beginner will be wise to use a cycloplegic frequently, because he has not had sufficient experience with all classes of patients to have developed that instinct which spots the " wrong 'un " at once. Nothing but constant work with all classes of individuals will give that intuition for the neurotic which suggests caution. The overworked mother, the daughter who has nursed her mother for three or four years before death, the head schoolmaster in an elementary school—these are the types who are liable to show an irritable accommodation, and who are most in need of an accurate correction. Then there is what I call the American temperament, and above all the man who has an acuity of $-\frac{6}{3}$. He must have an absolute correction, and it will be a very small one. If the glasses are ever so little inaccurate, he will at once suffer. I do not suggest that all these need a cycloplegic, but they call for caution.

I hope that I have shown no dogmatism and no special pleading. My wish is to elicit the truth, and I sincerely hope that there will be a discussion which will help to settle what I think to be a far more important problem than many we discuss in this room.

Prof. Gullstrand said he would like to point out some details of retinoscopy. If one used a silver mirror with a hole in it, a shadow was cast, which was a disadvantage in the examination. If the eye was without aberration, it ought to be a full shadow, but that was not so. The use of the silver mirror with a hole in it made the observer uncertain

as to what he saw. The only way to get rid of this inconvenience was to use a non-silvered mirror, *i. e.* a plate of glass with two plane faces. But then the source of light was not sufficient. The only source of light was a slit lamp, or the sun. If one used the slit lamp with this mirror, one could get refraction in the centre of the optical region of the eye and at the macula. By diminishing the slit one got the methods he had described in the third edition of Helmholtz's "Optics." The refraction could be measured in the central and peripheral parts of the tube. But that method was too sensitive for practical uses. But if one used a slit-lamp, without diminution of the slit, and with a plain non-silvered mirror, one got the best method for retinoscopy, and would never need to use a mydriatic. Mr. Harrison Butler said it was better to use a small hole. That was the right way, because if one had a hole it must be as small as possible, and the light source must also be as small as possible. Jackson, of Denver, was the first to point out the importance of using only a small source of light, and Dr. Woolf, of Berlin, was the second to do so. At the Washington Congress he, the speaker, showed a new modification of the slit lamp—a hole-lamp with a point lamp instead of the Nernst lamp, because a good Nernst filament lamp could not now be obtained, though one could buy an alleged Nernst. The lamps supplied by Zeiss used a current of $3\frac{1}{2}$ ampères, whereas the point lamp used only $\frac{1}{2}$ an ampère. Therefore the real method of determining a refraction without homatropine was a hole-diaphragm lamp with a plane mirror, not silvered—one with two parallel faces.

Mr. W. H. Brailey said the greatest difficulty in performing retinoscopy without a mydriatic was to get the patient to relax accommodation, and for the observer to be sure it was relaxed. A very valuable help was to take the manifest hypermetropia first—it occupied only a short time—and if the result was compatible with the final one, it was fairly certain to be correct: but if there was a large difference, he considered that a mydriatic was indicated. He considered that mydriatics were too much used in myopia: they were

seldom necessary. It was necessary to think of the patient and his normal life, which he was not going to live always under the influence of a mydriatic, and one should deal with it as a living problem rather than as a purely optical one.

Mr. Ernest Clarke acknowledged Mr. Harrison Butler's courtesy in sending him an advance copy of his paper. Knowing this contribution was to be brought before the Congress, he had made a number of observations and examinations. Mr. Butler said the main object was to give the sharpest vision, and make the patient comfortable. But there was one thing more. During the last 20 or 25 years oculists had recognised the meaning of eye-strain, and that a large number of patients came now with perfectly normal vision sent by a friend who had been cured in the same way, or by a physician. Taking 200 consecutive cases of refraction of all kinds, 77 of them had $\frac{6}{6}$ or $\frac{6}{5}$ in both eyes. That very much altered the aspect of the case. Mr. Harrison Butler said he had seen the result of the work of those who had used cycloplegics and of those who had not done so, and that they were both accurate. But who was the judge of that? Was Mr. Butler? Was there any criterion except the effect on the patient? Was the patient cured? "A" used cycloplegics. "B" did not. Were more patients cured by "A" than by "B"? With regard to the necessity for using a cycloplegic, in anticipation of the present discussion he took 100 consecutive cases and tested them carefully without a cycloplegic. Then he put them under atropine or homatropine, and he found that in 76 of them the result was different afterwards. Of the remaining 24, 21 were almost the same, but there was $\frac{1}{8}$ astigmatism of difference. Only 3 of the 100 were the same before and after the cycloplegic. He would go so far as to say that if 76 per cent. of the cases did not require a cycloplegic and 24 did, a cycloplegic should be used in a routine way so as not to miss the 24! Mr. Harrison Butler said spasm of accommodation was rare. The speaker admitted that the gross condition was rare. He had lately seen a boy who was wearing a -3, and under a mydriatic it turned out that he had hypermetropic astigmatism.

He was well after wearing a low plus cylinder. The 76 cases he referred to had had some spasm of accommodation; they had not given themselves up to the examination, and he did not see how one could be certain a patient was relaxing accommodation unless, in the majority of cases, he was put under a cycloplegic. If one was a careful examiner he did not think one need fear glaucoma. If he had a patient aged nearly 40 with much hypermetropia, he would not put him under a cycloplegic unless it became absolutely necessary. In the whole of his forty years of experience he had not had a case of glaucoma following the application of a cycloplegic. The dilated pupil, which Mr. Harrison Butler referred to as a disability, could be got over by using in the trial frame a disc with a central opening of 4, 5 or 6 mm., according to the distance of the disc from the eye. Mr. Butler also said it interfered with the celerity of his work if he had to put patients under homatropine. But that surely could be remedied by arranging one's work properly. As to the objection that the application of cycloplegics interfered with the patient's golf or tennis, if he preferred to go through life with headaches and to cultivate premature senility instead of going in for a cure of his astigmatism, that was his own look-out; but he should not be encouraged in that idea. He agreed that in the past atropine and homatropine had been used too much. If one once had in one's notebook a record of the static refraction of a patient, it might not be for years afterwards that a cycloplegic had to be used, and even then only in exceptional cases where there was a good deal of difference. What make of homatropine should one use? He had been advised to leave off using Burroughs Wellcome's Tabloid "W" because it caused a "stinging" of the conjunctiva. He had tried certain gelatine discs, which gave no pain. This was true, but they were not reliable, and he had therefore resumed using Burroughs Wellcome's tabloids, which were quite reliable. Drops he did not regard as reliable, because one did not know what proportion remained in the eye. He had now been doing refraction work for forty years, and he implored members not to be led away by the theories of Mr.

Harrison Butler. If they started on that idea, he was sure the result would be "sloppy" refraction work, and they would not cure their patients.

Mr. N. Bishop Harman said his views presented a mean between two extremes. Mr. Harrison Butler said that after he had used homatropine he found a lesser degree of hypermetropia than when he did retinoscopy without a mydriatic. The speaker thought that must have arisen from faulty observation as to the effects of the drug. Ten years ago the speaker did many refractions on the children in a certain school, using oily homatropine, under conditions which he had found very efficient. One day, when he was in a hurry, he examined the children sooner after the instillation than usual, and the first half-dozen he examined appeared to have zonular cataract. It was evident there was something abnormal in such a series of defects, and that the condition must be due to some defect in cycloplegia; therefore he put the children back to be examined later, and then he found the zonular appearance had gone. There was no doubt that as the mydriatic took effect it caused a gradual relaxation of the tension of the lens-fibres, so that there might be a bi-focal effect, or a circular line was showing between the periphery and the central part of the lens, and sometimes the appearance of a zonule. He suggested that when Mr. Harrison Butler made his observations showing hypermetropia less than the manifest he did the retinoscopy too soon after the instillation, so that the effect was incomplete. Under such conditions, possibly, mydriatics would be less reliable than observations without a mydriatic. If a series of observations were done from 15 to 40 minutes after the instillation, one could see the effect on the lens coming on. He was convinced of the value of the oily preparation of homatropine and cocaine as a cycloplegic in suitable cases.

Mr. Claud Worth said he hoped his refractions were not "sloppy": but for more than twenty years he had used, approximately, the methods which Mr. Harrison Butler had now described. He practically never used homatropine for purposes of refraction. For small children he used atropine,

but not on the same day, nor within an hour or two. For atropine to take effect reliably it should be used for three days. Homatropine he had found in many cases to be unreliable for refraction, and, except in the rarest instances, he had not used it for this purpose for years. One point mentioned by Mr. Harrison Butler was not stressed by that gentleman so much as it deserved. The point was, that one could not do a retinoscopy with undilated pupil on the patient's left eye with one's own right eye. He did not know the optical reason for it; but the part of the retina between the macula and the disc close to the macula gave almost exactly the same result in retinoscopy as retinoscopy on the macula itself; whereas an equal distance on the other side gave a very different result. In order to get the patient to relax his accommodation he had a dim light at the end of the room on a black surface; and therefore when the room was darkened there was nothing to be seen except this dim light, and when looking at that light the patient did not accommodate, and the dim light, after the momentary contraction of the pupil, ceased to cause any contraction. And one put one's head so that the patient was looking past one's right or left eye, as the case might be.

Mr. A. C. Brinton said he had the misfortune of working, in South Africa, at 6000 feet above sea-level, and there he found his patients suffering from asthenopia of high degree in consequence of a very small degree of refractive error. That was the backbone of the ophthalmologist's work in that district (Johannesburg), as refractions were to those here. His partner, Dr. Napier, and he had come to the conclusion that Mr. Ernest Clarke was one of the very few who did their refractions sufficiently well to suit the altitude he, the speaker, referred to. When they got a refraction done by a Scotsman, they knew it would be wrong before starting! Dr. Napier and he believed that the reason was that they had a very bright light, and the ciliary muscle became very tired from working in such a bright light at that altitude. He had played golf for many years, but he got tired after 36 holes in Johannesburg. Here, in England,

however, he felt he could do another round. The effect on all the muscles was the same, so that the apex-beat in many of the school children had been found to be outside the nipple line; and cross-country racing for children had had to be stopped. Those present could appreciate the effect on the muscles when it was a slight error, and the error of refraction was a strain during all the waking hours. He analysed a few hundreds of cases of headache, and had intended to read a paper on them before the Congress, but he did not intimate his wish soon enough. He found that 37 per cent. of his asthenopic cases took ·25. He often had to refract ·13 to give comfort. The astigmatic error he was speaking of was only ·5 per cent. He had seen exaggerated symptoms, such as vomiting and a simulation of sunstroke, from these small errors in or about the horizontal position. During the German East Africa campaign many of the soldiers were sent to Cape Town, where he was on active service. They were sent with the diagnosis "sunstroke," yet they were mostly men who had been sent from home and were not accustomed to the glare. In every case they had astigmatism against the rule, somewhere about 180: therefore he got all the medical officers in the hospital to send him their cases of "sunstroke," so that he could examine their refraction, even if they had not come to his particular department in the first place. In all these cases his partner and he made it a rule to use a mydriatic. Most of the men he was referring to were first-class marksmen; there was nothing the matter with their sight, and if mydriatics were not used the patient came back subsequently with a headache. He learned to use a mydriatic in Birmingham Eye Hospital, before Mr. Harrison Butler went there, and he had adhered to the practice. When he was in Philadelphia Prof. Fox said to him: "It is a pity so many good English eye men do their refractions so badly."

Mr. A. Hugh Thompson said Mr. Harrison Butler had emphasised the importance of doing refraction at the macula, but he did not explain how it was possible to do that when the patient was looking to the side of the examiner's head

at an object at the other end of the room. Mr. Worth had hinted that it did not matter, because the refraction between the macula and the disc was constant. But the speaker suggested that this did not hold—at any rate in cases of myopia. He hoped to hear the point dealt with in Mr. Harrison Butler's reply.

Mr. Traquair said that in Edinburgh ophthalmologists had the distinction of being unanimous in their attitude towards this question. They thought, there, that this use of cycloplegics was a matter for determination; that there were cases in which cycloplegics were necessary, while there were others in which a good correction—one which satisfied the patient and removed his headaches—could be secured without using a cycloplegic. The Edinburgh practice was to use a cycloplegic for children, but not for adults. Even in the case of children it was not done on account of fear of spasm of accommodation, or of latent hypermetropia, but because it was difficult to make a child look steadily in the required direction, and for similar reasons. Under such circumstances it was much easier to do retinoscopy with a dilated pupil and paralysed accommodation. Instead of making retinoscopy the final criterion they were inclined to lay the greatest stress on accurate subjective examination. If an optician had ordered a -3 cylinder for a case of hypermetropia, the error was to be attributed rather to the fact that the optician did not understand subjective testing than to the absence of cycloplegia. At Edinburgh they thought it was possible to discriminate between those cases in which a cycloplegic ought to be used and those in which it was not necessary. And when one did pick out these cases, which were not very numerous, in only a few was there any real difference between the error found with a cycloplegic and that found without it. With regard to doing the retinoscopy in a dark room, he liked, in hospital, to leave the door open, so that the patient could see to the end of a long corridor, and he asked the patient to look at an object at the end of that passage. That enabled the accommodation to be relaxed. In his own room he used a little picture at the other end of the room, 20 ft.

away, which served to attain the same object. In reply to Mr. Harrison Butler's question concerning the ophthalmometer, he thought that in order to use that instrument to find the cylinder required to relieve the patient's symptoms, it was not necessary to have cycloplegia, and that from the retinoscopic and ophthalmometric examination without cycloplegia and a careful subjective test, upon which he laid the greatest stress, the cylinder could be estimated quite well. They found the ophthalmometer of enormous use, and on that point also the Edinburgh Infirmary Ophthalmic Staff were unanimous. With reference to subjective testing, especially in hypermetropia, after the correction of each eye singly equal plus spheres should be added with both eyes open. By this procedure sometimes as much as 2 D. of *plus* sphere would be accepted over the maximum which had been accepted with each eye singly. In that and other ways the under-correction of hypermetropia could be avoided. The aim should be to prescribe glasses which the patient would wear with comfort and would see with at a distance. In order to achieve that object under the conditions of cycloplegia it was necessary to have a post-cycloplegic test: and if that was needed, why not begin with it? With regard to the accuracy of refractions which had been referred to, if they were to test by retinoscopy to ·125 D., they were all likely to find each other's refractions inaccurate, for it was difficult for any two practitioners to find themselves in harmony to the extent of only $\frac{1}{8}$ D. And while he had experienced the benefits of prescribing for the correction of such small errors, there was always a doubt in his mind whether the advantages obtained were not frequently due to subjective impressions derived from the consultation, which have a powerful influence on some patients. He had a letter from a medical student, for instance, stating that the use of a pair of $-0{\cdot}25$ D. spheres had cured his asthenopia, which had prevented him from studying. In that case no cycloplegic had been used.

Mr. Stack expressed his agreement with everything Mr. Traquair had just said. He would like to emphasise every

point in those remarks, and especially the policy of the "ever open door," particularly in hospitals, where the examinations were sometimes shut in by a curtain. It was most important for the patient to be able to see in the distance. Mr. Harrison Butler had done the same in his excellent room, which the speaker had seen; there was a distance of 25 to 30 ft. to his red and white discs. There was one reason for giving a cycloplegic which Mr. Harrison Butler had not mentioned, namely, where one found an error of hyperphoria. Before ordering a prism, up or down, one liked to know that the patient still had hyperphoria when under the cycloplegic.

The PRESIDENT said the discussion had been a very interesting one. Members were really seeking the absolute where, possibly, there was no absolute, and there were, as it must be admitted, many different ways of determining the refraction of the eye. It would not be possible for this body, nor for any other body, to settle for everybody which method gave the absolute and perfect result. Information was gleaned from all methods, and it was by the correlation of all methods in each individual case that the best ultimate result would be arrived at. But it was not possible to use every method to determine the refraction in every instance. Some members of the ophthalmic profession developed a special capacity in one direction, others in another direction, and for any one man to be coerced into adopting other men's methods would destroy the utility of his work. But what would probably be more enlightening than trying to arrive at one absolute method would be to consider the imperfections which arose in the various methods, all of which were valuable. But for the restriction of time, the debate might have usefully gone on further yet.

Mr. HARRISON BUTLER, in reply, expressed his pleasure at hearing the views of Prof. Gulstrand. They had all sat at his feet for many years, but he feared that few of them would ever reach beyond the ankles. He would like to try retinoscopy with the slit lamp, but for the present the excessive cost of the apparatus deprived him of this experience. Mr. Ernest Clarke had gone into the matter and he had found

differences between his results with and without atropine. He was not surprised to hear it, but the question was which was the correct refraction. With regards to difference of $\frac{1}{8}$ D., there were a few individuals with an acuity of $\frac{6}{3}$, and these could often with certainty choose between two lenses with a difference between them of $\frac{1}{8}$. But this $\frac{1}{8}$ difference generally varied from time to time, from month to month, and perhaps from day to day. He had had the opportunity of going over many of Mr. Ernest Clarke's cases, and he had occasionally found that he could improve them by a very slight correction. This might easily be due to transient alterations, which he was sure amounted to at least $\frac{1}{8}$ D. He wished most emphatically to emphasise that his methods were not sloppy: he deprecated "sloppy" refraction as much as Mr. Clarke. He considered that it was not necessary to slop in atropine in the eyes of all and sundry, but only in selected cases. He devoted at least half an hour to every refraction, and he did not think that a private case could be worked out in much less time. He agreed that glaucoma was not to be feared from homatropine if reasonable precautions were taken. The tension must be estimated and the discs examined before using a mydriatic. He could confirm Mr. Bishop Harman's remarks upon the futility of refracting before the cycloplegic had had time to act fully. Under partial cycloplegia the result might be wholly inaccurate. He agreed with Mr. Worth regarding the time necessary for full atropine cycloplegia in children. For convenience sake at his school clinic the atropine was instilled for an hour only. If he arrived a little too soon and the atropine had not had the full time, he found many cases in which refraction was impossible. He believed that it was far better in children who needed a cycloplegic to instil it for two or three days. He was pleased to hear what Mr. Brinton had said. He had practised in South Africa, and was aware of the conditions. He did not doubt that the American temperament was more common in Johannesburg than in London, and that people fiddled and fussed more with their refractions than in this country. He realised that there were men with $\frac{6}{3}$ acuity

who could discriminate the value of a $\frac{1}{8}$ cylinder, and some of them were made happy by such a minute correction. He also found that a plus cylinder horizontal was often called for when a similar vertical error caused no symptoms. Answering Mr. Traquair as to the inattention of children, if working without a cycloplegic, it was essential to examine in a quiet room, and, if possible, alone. The examination could not be done in a crowd or in the presence of anything liable to diminish the child's power of concentration. In answer to Mr. Hugh Thomson: He was aware that refraction without a cycloplegic was not exactly upon the macula, and this might account for many of the discrepancies between the retinoscopy and the patient's choice. The final act of all refraction was with the trial frame: in the end the patient had to chose his own glass! Mr. Priestley Smith had pointed out the fact alluded to by Mr. Traquair. Often a patient would take more plus with both eyes together than with either alone, but this was not an invariable occurrence; in fact, it was the exception rather than the rule. In a recent case he had worked up to 1·5 D. by this method, following retinoscopy, which gave 1 D. He thought that there might be some latent hyperopia and instilled homatropine for an hour. Under the cycloplegic the lady would not take more than $\frac{1}{2}$ D.—that is to say, the cycloplegic result gave 1 D. less hypermetropia. He had had scores of similar cases. Regarding hyperphoria, mentioned by Mr. Stack, he thought that if it existed in sufficient degree to warrant correction with prisms, then it would be obvious without a cycloplegic.

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2. *On the ætiology and prevention of convergent concomitant squint.*

By Percival J. Hay.

When the following records were begun, it had been my impression, almost amounting to a conviction, that certain factors in the personal history of squinting children played

a much more important part in the causation of squint than they had up to then been given credit for. And so, without putting any leading questions, the parents were asked in every case to what cause or circumstance they attributed the squint.

TABLE I.

*Group A.**

Measles	130
Birth or early infancy	47
Whooping-cough	22
Entry into school	11
Teething	11
Scarlet fever	4
Broncho-pneumonia	24
Convulsions	15
Influenza	10
Falls and other injuries	37
Other causes	32
	343

Group B.†

No ascertainable cause	254
	597

Patients with relatives who squint.‡

Of Group A	43	have such relatives	
Of Group B	57	„	„ „
Total	100		

* For reasons of economy the details of these tables, which were shown to the Society, have been omitted.

† Many of the cases under this heading are probably attributable to measles.

‡ In many of these relatives measles or some other affection will have preceded the squint, so that heredity probably plays only a minor part in the causation of squint.

The total number of cases investigated is 597. Of these a history was obtained in 343. About the remaining 254 no information could be gathered, either because the parent had forgotten or had not noticed any apparent cause, or because the child was accompanied by an elder brother or sister, neighbour or foster-parent who was unacquainted with the history of the patient.

Table I shows the total number of cases and their distribution. In group A, comprising those cases in which a history could be obtained, measles and scarlet fever present the most striking figures, viz. 130 and 4. This great difference must be accounted for. It is clear that in the case of scarlet fever we might discount the parents' statements, because the number is so small as to come within the region of coincidence or mistaken observation ; but when the figure is so large, as in the case of measles, we are bound to accept the situation, and to say that the evidence must be, on the whole, trustworthy. It is obvious also that the parents can have no interest in attributing to measles what they might as well, if they felt so inclined, attribute to scarlet fever, both being common diseases of childhood. It might be argued that measles is more common than scarlet fever, but the incidence of measles as compared with scarlet fever is only about 7 to 1, whereas the incidence of squint following measles as compared with squint following scarlet fever is from our figures 30 to 1.

The theory that hypermetropia has caused these squints falls to the ground, because we cannot imagine that hypermetropia is reserved for children subject to measles, and that scarlet fever cases are emmetropic or myopic. Nor can we suppose that the fusion sense is less well developed in children who are going to get measles than in those who are going to get scarlet fever. We must seek for another explanation, and I conclude that measles in itself contains the cause of squint. It is well known that measles is often anything but a slight disorder, and, apart from having a death-rate that is considerable, it can also cause severe nerve lesions, such as polyneuritis, paraplegia and hemiplegia. Incidentally, it will be remembered that it is the only one among the exanthemata

of childhood which regularly affects the eyes and causes an intense conjunctivitis. I think it is reasonable to assume that measles, and perhaps other diseases, such as whooping-cough and influenza, which can act upon the nervous system, produce a toxin which in some cases temporarily or permanently affects the nervous mechanism of which the sixth nerve forms a part.

I am not very much concerned in propounding a theory as to how and where the toxin acts, but perhaps some speculation might be allowed. The impairment of movement always concerns both external recti, so the lesion is necessarily a central one. We know that the nervous control over the action of the external rectus is a double one, from the fact that in conjugate deviation of the eyes, paralysis of the lateral movement occurs, without any interference with the act of convergence and divergence, and we know that the opposite is also true.

It may be supposed, therefore, that there are two centres governing the movement of the external rectus, one of them concerned in controlling the conjugate movements of the external rectus of one side with the internal rectus of the opposite side, and the other a centre for divergence concerned in presiding over the associated movements of the two external recti, in balancing the movements of the interni in the act of convergence and divergence. A mere lack of tone in this centre would probably just be sufficient to allow the eyes to overbalance, if I may use the expression, and a more severe interference with the functions of the centre would account for greater deviation.

On this assumption many clinical facts and observations receive an explanation. First of all, we can understand why a squint may occur where there is no error of refraction: also where the refraction is low and equal in amount. We can also see why some cases of nebulæ squint and others do not; why some cases of congenital cataract squint and others do not; why only a small proportion of the population afflicted with hypermetropia squint and the remainder do not; why, on the other hand, a squint may be absent where

there is a unilateral high degree of hypermetropia with very defective vision, the patient not having been affected by one of the exanthemata or other causes under review. It explains why there is no relationship between the amount of refraction and the degree of deviation. It accounts for the fact that some cases recover spontaneously without treatment, some quickly and some only after a lapse of time. It explains why correction of the refractive error, which diminishes the impulse required for accommodation and convergence, is sometimes all that is necessary to restore balance between convergence and divergence, and it explains why the instillation of a mydriatic has the same effect.

This centre, however, is not only sensitive to a toxin, such as is produced by measles, and to a lesser extent perhaps by influenza and other affections, but also to injury, and I think that the statements made by parents who attribute the squint to a fall, or to convulsions, or to the strain of whooping-cough, cannot but be accepted in the majority of instances; probably the cases of squint following birth of the child are of similar origin, and one cannot but think that a proportion of those that are attributed to "entry into school" are not due to the fact that the child is more occupied with close work, but rather to the fact that the child in its new surroundings gets knocked about, falls, receives blows on the head from its school-fellows, etc., and so the condition is really one of injury. And while I think that perhaps further evidence is required, it is quite possible that physical or mental distress or shock, such as we find in teething, burns, and severe operations, may also act upon this centre and cause a squint, as a result, perhaps, of bio-chemical changes.

The exciting factors in the causation of squint might be tabulated as follows:—

TABLE II.—*Exciting Factors in the Causation of Squint.*

(1) Toxæmia—measles, broncho-pneumonia, whooping-cough, influenza.

(2) Trauma—school games, falls and other injuries, convulsions, whooping-cough, birth.
(3) Physical and mental distress or shock (?)—teething, burns, operations, etc.
(4) Imitation (?).

A share in the production of squint has been claimed for many other conditions. Without appraising their merits, I should feel inclined to classify them as predisposing causes, so that finally all the factors in the causation of squint might be conveniently grouped under the following headings :

TABLE III.—*Factors in the Causation of Squint.*

1. *Exciting factors :*
   (1) Toxæmia.
   (2) Trauma.
   (3) Physical or mental distress or shock ?
   (4) Imitation ?
2. *Predisposing factors :*
   (1) Hypermetropia (Donders).
   (2) Defective fusion faculty (Worth).
   (3) Convergence excess.
   (4) Divergence insufficiency.
   (5) Hyperphoria.
   (6) Heredity.
3. *Developmental factors :*
   (1) Inherited.
   (2) Not inherited.

" Prevention is better than cure." I have already stated that it is not a matter of very great moment how measles causes squint, but the circumstance that measles is an important cause of squint must, if we are satisfied on the point, be of general concern. For although we cannot prevent patients from getting measles and so from getting squint, if it were generally and sufficiently well known that measles is responsible for a large proportion of the cases of squint, the practitioner would be able to impress the fact upon the

parents, and to warn them that they should be on the look-out for this complication, and to instruct them to bring the child at once for advice. There is no doubt that the longer the imbalance prevails, the more difficult does it become to restore it by such simple means as correction of the refractive error and the instillation of a mydriatic. On the other hand, I am convinced that the establishment of a squint could be prevented in many cases if the child were brought as soon as the squint was noticed. We cannot hope that this will be done until the general practitioner is made aware of the fact that squint is a complication of measles and sometimes of the other exanthemata. At present he does not know, because the squint does not occur during the time of his attendance—which generally lasts only a week—but occurs two or three weeks afterwards, and sometimes later.

Mr. Claud Worth said members had listened with great interest to Mr. Hay's paper, and the tables shown were valuable and useful additions to evidence. In the main he agreed with what Mr. Hay had said, though there were several minor points of difference. In hospital practice the speaker had carefully noted down the reasons to which the squint was attributed by the parents. Mr. Hay found that measles was blamed for it about six times as often as was whooping-cough. In his own cases at Moorfields the blame had been distributed nearly equally between measles and whooping-cough. Another curious point: In private practice one asked the mother to what she attributed the squint; also when she first noticed it. One expected to get a history from these people more often than from hospital patients, but in private practice one found the squint attributed to some infantile disease much more seldom, which leads one to think that hospital patients often draw upon their imagination. Mr. Hay said one would not expect that patients with defective fusion sense were more likely to get measles, and the speaker agreed; but patients with defective fusion sense getting measles were more likely to squint. This was shown by the fact that a patient who had had measles and who had

recently begun to squint did not see double, whereas a child who, from diphtheria, had a squint, manifestly of local origin, saw double, and that was a very clear point. As to predisposing and immediate exciting causes, it was not a very big point as to whether one called a particular factor an exciting cause or a predisposing cause: it was difficult to separate them. He, Mr. Worth, maintained that in a typical case of squint this defect of the fusion sense, this want of the potential faculty of binocular vision, was the cause which made the squint possible: and anything which upset the convergence, whether by means of the nerve muscle mechanisms or through the accommodation in any way, was likely to cause squint.

Mr. Maddox said he was interested in Mr. Hay's paper. It would seem as though any depletion of the nerve energy of the oculo-motor apparatus would naturally affect most the weakest conjugate innervation, namely, that concerned with divergence. Measles is, perhaps, a powerful depleting cause. It was a valuable thought the reader had brought forward, namely, in the ætiology of squint to take the divergent reflexes more into account. So weak were these that many doubted their existence. Sir George Berry, for instance, would not listen to the suggestion that there was a divergent innervation at all. For many years the speaker thought it most likely that it was connected with the sympathetic nervous system acting in antagonism to convergence in the same way as the dilating fibres of the iris were antagonistic to the sphincters. In children convergence was very powerful, and the diverging faculty was the one most prone to suffer from any nervous depletion. He agreed with Mr. Hay that it would be the central conjugate diverging reflex which was affected, not merely the sixth nerves.

Mr. Pickard said this question was partly a matter of simple statistics. He did not deny the possibility that the contention of the reader of the paper was true. It might be that his position was stronger than was apparent, but he thought the evidence brought forward was not complete. It was not complete on the question of the incidence of these

diseases or causes in the general population. For instance, the incidence should be stated in the particular specimen of the population one had to deal with in that particular locality, at that age, and the amount of measles existing at that time. Unless such figures were available for comparison with the cases under consideration, the evidence was not complete. The same might be said of the evidence of injuries to children. Until these details could be brought forward, the question could not be regarded as proved.

The PRESIDENT said the last speaker's remarks were quite to the point, as, no doubt, Mr. Hay would agree. Statistics were notoriously misleading. When the evidence was complete, it should be scientifically sifted and the facts correlated by a trained statistician. Usually all that was done when people used statistics, as they believed, was that they abused them.

Mr. HAY, in reply, thanked the speakers for their remarks. He added that the cases had been collected over a period of six years, and that they had been drawn from a population exceeding 1,000,000, so that he did not think that any one of the exanthemata was likely to show an undue influence in the tables.

VIII. MISCELLANEOUS.

1. "*Delhi-green*" *glass.*

By N. Bishop Harman.

Some years ago I had presented to me an old pair of tortoise-shell spectacles. Attached to them was a label inscribed, "These spectacles were found at Delhi, India, just after the Mutiny. The green lenses are a most perfect neutral and cannot be produced to-day." The spectacles are believed to be of European manufacture, but where the glass contained in the rims originally came from cannot be judged. I must confess that on reading the inscription my impression was one of incredulity so far as the qualities of the glass were concerned, but later I found that the statements were in fact true.

The comfort of the glass for use in brilliant sunlight was unmistakable, and, desiring to get some like them, I tried one or two firms without success. Messrs. Hamblin's, of London, said they would try to secure a match, and after much trouble on their part they have succeeded in getting very close to it. The features of the glass are these: The green tint is comfortable and does not make everything of too violent a green hue: there is sufficient transmission of light to give some colour sense: it is therefore cheerful. When tested for its spectrum, it is found to give a better reduction of both the red and ultra-violet ends, combined with a high percentage of transmission than any other glass in common use. I have given it the name of "Delhi-green" glass for convenience in reference.

Spectrum photographs have been taken with a number of glasses in common use by S. Judd Lewis, D.Sc., and these

were shown in illustration; also a graph has been prepared showing both the effective wave lengths and transmission of these different glasses of equal thickness (2 mm.).

A nickel-iron spark gives a very long spectrum, from the ultra red of over 7000 wave length to an ultra-violet of some 2150. The interposition of these glasses between the spark and the films gives the following effects:

White spectacle crown causes no change at the red end, but shortening of the ultra-violet end to about 2950, with a small loss of transmission.

London smoke has very little more effect upon the spectrum than the foregoing; it has practically no effect on the red end and only slightly shortens the ultra-violet end. But transmission is reduced to about 55 per cent. It is an amazing experiment to put up a dense black piece of London smoke glass and to find that it has apparently no effect in checking the passage of the rays of a mercury vapour lamp.

Signal green, or what is commonly known as *peacock-blue*, has most effect at the red end, relatively less at the ultra-violet end, but it reduces transmission considerably to nearly 50 per cent.

Pot blue, in common use amongst furnace workers, gives a very limited wave-length to the blue, violet and ultra-violet; it has not much effect at the ultra-violet end, but its visibility is good for blue, being 80 per cent., but since its spectrum is so limited it is a very dark glass.

Crookes' A gives excellent transmission—some 88 per cent. It is ineffective at the red end; but more effective than the pot blue, and about equal to signal green, at the ultra-violet end.

"*Delhi-green*" cuts off the extreme red end and all the ultra-violet rays, which Crookes' A does not. Its transmission is good, 65 per cent.—that is, better than signal green, or London smoke, and coming close after Crookes' A.

These spectrum and transmission effects confirm the feeling of comfort which the wearing of this glass produces, and I suggest that this is the type of glass which will prove most comfortable to our patients who need protection from exces-

sive glare when out of doors either in this country during very bright weather or in the tropics, or for patients suffering from disease conditions that demand protection from glare. We need a glass which combines a pleasant diminution of the intensity of the light, yet allowing for what I have called visibility, combined with complete protection from the hot red rays and the irritating ultra-violet rays, and the "Delhi-green" glass gives this.

Mr. Gardner (ex-President, Röntgen Society) said he was extremely pleased to have witnessed this exhibition of Delhi glass. He spent the greater part of his life with the late Sir William Crookes, and at the date of his death they were in the midst of experiments in the production of a glass which would be more suitable for these purposes. The work was still going on, though, he was sorry to say, very slowly. The glass now shown had a very peculiar absorption band in the middle of the green, and he hoped he would have the opportunity of examining it more closely. The aim of Sir William Crookes was always to produce as much light as possible—ordinary visible light—while cutting off ultra-violet and heat rays. That aim was still before those who were working at the problem. The absorption of the glass shown was extremely interesting. He did not understand from Mr. Harman whether he succeeded in getting it reproduced in England; but if there was any difficulty about that, he felt sure, from the position he was in now, that he would be able to reproduce it without difficulty. He said that the demand for a glass transmitting blue and green was much greater than for a glass which cut off the greater part of the infra-red rays. The latter was being produced in only very small quantities in consequence. The price he knew nothing about; he was interested only in the production.

Sir John Parsons said Sir William Crookes entered into a scientific investigation of various kinds of glass, introducing into them various metallic salts in his efforts to discover a glass which would cut off both infra-red and ultra-violet rays. It was very difficult to obtain a glass which did both without

producing a very considerable reduction in the transmission of light. There were several glasses, possessing different properties, which were selected out of some hundreds which he made, those having the best properties for certain purposes. There was at present a boom in the elimination of ultra-violet light for clinical purposes, and it was only natural that those particular glasses which were known as " Crookes' A" and " Crookes' B " should have got on the market, as they transmitted the maximum amount of light and eliminated the maximum amount of ultra-violet radiation. They were not made specifically for the glass-blower at all, because at the outset of the investigation by the Royal Society Committee it became improbable that glass-workers' cataract was due to ultra-violet radiation. With regard to heat absorption and the question of the British working man, which Mr. Butler made striking reference to, and to the Government action in the matter, those remarks were not quite justified. Messrs. Nuttall, at St. Helens, were kind enough to place their work at the disposal of the Glass Research Committee for investigation ; also that firm got a considerable number of goggles containing various glasses, which were placed at the disposal of the workmen, and they were urged to use them. As a result, however, the workmen would not use these glasses. With regard to acetylene welding, the Royal Society Committee had nothing to do with that, although some of Sir William Crookes' glasses might have been suitable. The British worker was under no disability in regard to that, because he could not work at this welding unless he wore goggles. The Departmental Committee on Blindness were seriously concerned about the question of wearing goggles. It was a very important point economically, particularly the wearing of protective goggles against the entry of foreign bodies. Most people would agree that the loss economically from loss of time and the payment of compensation far exceeded the amount paid as compensation, etc., in miners' nystagmus, which was said to be about a million pounds a year. Yet, however much persuasion was tried, it was impossible to get the British working man to wear goggles

at his work; it was considered by him "bad form," and hence was taboo. Some regarded it as effeminate. Even penal measures did not lead to their adoption. For visual purposes the Delhi-green glass might be very comfortable, but he did not think that, for ordinary purposes, it was the ideal. It absorbed an enormous proportion of light, and if people wore glasses tinted with colour of any kind, it meant they became adapted to the particular colour. He thought the ideal, from the purely visual point of view, was to get a glass which absorbed the maximum of the ultra-violet and a considerable quantity of the heat rays. If a glass absorbed any great quantity of the heat rays, it became in itself a source for the radiation of heat, *i. e.* it became hot, and itself radiated heat. With regard to the luminous part of the spectrum, what was wanted was a glass which would diminish the intensity of the light evenly throughout the visible spectrum, without any definite absorption bands at all. A committee which sat during the war considered the question of smoked and otherwise darkened glasses for the soldiers in Mesopotamia, and on that Committee Mr. Chance, physicists, and others were engaged. At that Committee he, Sir John, asked whether it was possible to obtain a tinted glass which would simply diminish the amount of light evenly throughout the spectrum. Everyone agreed that this was practically impossible. All the tinted glasses possessed absorption bands. At the same time, the glasses tinted with London smoke absorbed fairly evenly through the length of the spectrum, and it did not mean a special colour adaptation, which, he thought, must be detrimental for certain purposes; it was an abnormal condition.

Mr. Healy considered the question now under discussion a very big one, and that it was worthy of a full discussion on some future occasion. With regard to the use of goggles for protective purposes, especially against the entrance of foreign bodies into the eyes, that was a question in which he had tried to take an interest in the various works in his district; and he found it very difficult to get workmen interested, especially the older ones. He was, however,

tackling the younger men, especially the better educated among them, such as those who were attending the welfare centres of the works. Most big works nowadays had these centres, and had very good men at the head of them. Much good, he thought, could be done in that way. He had got some of the younger men to adopt goggles ; he used ordinary triplex glasses. Much could be done by getting into touch with some such organisation as the " Safety First League," which could arrange lectures where spectacles could be demonstrated with the markings made on the glass by the foreign bodies. He thought all ophthalmic surgeons who were working in industrial districts should try to organise popular lectures on the subject. It would require a long time to get the working man to realise the importance of it, but the prejudice on the subject would, he believed, be overcome by tackling the younger men ; it seemed hopeless to make any attempt to impress it on the older ones. With regard to the question of the visibility of various protective goggles from heat rays or ultra-violet rays, he had been trying to use Crookes' glass for that purpose, but steel smelters used largely non-standard cobalt blue. He had tried to get them to adopt the very deep Crookes, cutting off the ultra-violet rays. Those who had tried them admitted they were more comfortable and that they saw better with them, but that there was a difference in the visibility, and they could not so well judge with them the exact time at which to tap the furnace. It was a question of long experience with one type of glass.

The PRESIDENT said the discussion must not be allowed to extend to the subject of mechanical protection of eyes from injury, though the Society was glad to have attention drawn to that, because one knew the great loss of time and the amount of injury which resulted from the lack of such protection. And Mr. Healy's suggestion on that point was useful, as it might lead to surgeons spreading the idea more widely. Everyone knew the difficulty of getting protection, and he was impressed by the enormous number of people who were suffering from foreign bodies having become impacted in the

cornea, especially during the sharpening of tools at emery wheels. He himself took steps to communicate with Mr. Lloyd George when he was Minister of Munitions, and asked whether it would not be possible to increase the output of war munitions by the provision of protecting goggles when working at emery wheels. But even at that time and in those circumstances the whole thing failed; whether owing to inertia in the Government Department or because they were aware of the impossibility to get men to wear goggles he did not know. Valuable suggestions had now been made, and perhaps the matter might be furthered on the lines suggested whenever there was an opportunity.

Mr. Stack asked whether there was any method for ascertaining whether a certain glass was really Crookes' glass or not. The public had got the idea of the Crookes' glass, and many were insisting on having the Crookes' "A". Patients came to him with spectacles which they had given a lot of money for and told him they were of the Crookes' glass. Crookes' were a purple tint, but he did not know whether there was an easier method of making sure. If the glass was in a frame, he at present had to take it out and get it into a particular light, and test it against white paper in order to bring out the purple tinge one saw coming through the edge of Crookes' glass. He had several times demonstrated that the patients who considered they had Crookes' glass had not that kind at all.

Mr. Bishop Harman, in reply, said the glass was not considered perfect, and he would be delighted to make it better. The particular specimen he showed was made by Chance, of Birmingham. It was not an industrial glass, and was not intended for any and every patient—certainly not in this country. Here the average patient wanted a glass as white as it could be got. This glass was for those who were subject to glare, here, but especially in the Tropics. Patients, under certain circumstances, liked a tinted glass; they were not content with glasses which were nearly white; they liked something which reduced the sense of light to their eyes. And it was particularly for those who suffered from

some defect of vision, such as arose from disease, cyclitis or retinitis: these patients wanted special protection—they liked this glass. It might not be scientifically perfect, but it was a glass that was very comfortable to wear.

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2. *The aqueous humour and novarsenobenzol. An experimental investigation into the composition of the aqueous humour and some of the organs of rabbits, after intravenous injection of novarsenobenzol.**

By Humphrey Neame and Frank W. Bury, M.Sc.

Object.—The object of this investigation was to try and establish whether any arsenic was passed into the anterior chamber after intravenous injection of Novarsenobenzol in rabbits.

(1) *Inoculation of novarsenobenzol and collection of aqueous humour and organs.*—Three rabbits, Nos. 38, 39 and 40, were given each an injection into the ear vein of 0·3 grm. of novarsenobenzol, batch No. 102 of Corbière, Paris, dissolved in 5 c.c. of saline. Two hours after the time of injection the rabbits were killed instantaneously by intravenous injection of 10 c.c. of air into the same vein of the other ear. Within ten minutes of death the aqueous humour was withdrawn by means of a long graduated 1 c.c. syringe fitted with a half-inch needle of the smallest calibre obtainable. The fluid was withdrawn without draining the anterior chambers dry, so as to avoid the likelihood of suction of blood or serum from the iris. In all, five out of the six eyes were so tapped. It was necessary to mix the five samples of aqueous in order to obtain a quantity of sufficient size for analysis.

Heart blood, liver and muscle were taken from No. 38 rabbit, for comparison.

The weights of the rabbits were as follows:—

* The writer records his acknowledgments to the Yarrow Research Fund of the London Hospital, by whose generosity the expenses of this investigation were defrayed.

No. 38	3,050 grm.
No. 39	4.010 ,,
No. 40	3,030 ,,
Total . . .	10,090 grm.

Total novarsenobenzol injected in the rabbits, 0·9 grm.

Average proportion of novarsenobenzol is 1 part per 11,210 parts of body-weight.

This is a fair comparison against which to put the arsenic content of the anterior chamber, but not quite fair for the arsenic content of the organs, for the latter were all taken from No. 38 rabbit. For practical purposes it is sufficiently accurate, its error being not more than about 0·1 per cent.

The weights of the various samples are their weights as taken immediately after they were bottled, before any evaporation had occurred. They were calculated in the usual way, and obtained by subtracting the weight of the empty glass vessel and stopper from the weight of the same occupied with the respective tissue :

Aqueous humour . . .	1·86 grm.
Blood	6·15 ,,
Liver	27·4 ,,
Muscle	26·25 ,,

(2) The following is the report by Mr. F. W. Bury, M.Sc., who kindly undertook the very laborious analysis of the above tissues :

Report on the Amount of Arsenic found in the Liver, Blood, Muscle and Aqueous Humour supplied.

Method of analysis.—The method adopted for testing the organic matter was the one prescribed in *The Standard Methods of Chemical Analysis*, by Scott. The finely chopped and well-mixed sample was placed in a large flask and enough water added to produce a fluid mass.

An equal quantity of concentrated hydrochloric acid and 1 to 2 grm. of potassium chlorate were added, the flask shaken and placed on a steam bath. Nascent chlorine is evolved, which vigorously attacks the organic matter :

0·5 grm. portions of potassium chlorate are added at half-minute intervals.

When the organic matter had decomposed, the mass was diluted with water and filtered. The arsenic is found in the filtrate.

The filtrate was made up to 500 c.c. in the case of liver, muscle and blood.

The aqueous humour solution was evaporated down to dryness to expel excess acids and decompose the chlorates, and then made up to 100 c.c. with water.

Ten c.c. of this solution were added to the Marsh apparatus, but no mirror was produced, so that further portions were added until the whole of the 100 c.c. was used. *No arsenic was found in the aqueous humour.*

Fifty c.c. of the filtrate from the liver, muscle and blood were evaporated down to dryness and made up to 100 c.c. with water.

Measured portions of these solutions were added to the Marsh apparatus until a mirror was obtained, which came within the scope of the standard mirrors already prepared.

Standard mirrors were prepared corresponding to ·003, ·005, ·007, ·009 mgrm. of arsenious oxide.

Typical analyses.—Quoted from the paper of William Henry Wilcox, M.D.Lond., B.Sc., F.R.C.P., and John Webster, F.I.C., published in the *British Medical Journal*, 1916, vol. i, p. 478.

Dog, weight 24 lb. 3 oz. Neo-salvarsan 0·3 grm. was given and the animal killed 24 hours later. Specimens kindly sent by Dr. Macintosh and Dr. Fildes.

Liver . . .	1·0 mgrm. of As_2O_3 per 100 grm.
Kidney . .	0·4
Muscle . . .	0·08
Blood . . .	0·1
Brain . . .	Trace only
Bone . . .	Trace only

Result obtained in samples supplied :

Liver.—0·82 mgrm. As_2O_3 per 100 grm., or 1 part per 120,000.

Blood (whole blood).—1·2 mgrm. As_2O_3 per 100 grm., or 1 part in 83,333 parts.

Muscle.—0·08 mgrm. As_2O_3 per 100 grm., or 1 part per 1,250,000.

Aqueous humour.—None.

The PRESIDENT said all were interested in the treatment by these arsenic compounds, and therefore all knowledge obtainable about them was of considerable importance. The present communication broke the ice somewhat. His own ideas on the treatment of syphilitic diseases of the eye had led him to think that the present position was, on the whole, somewhat unsatisfactory. They had been taught to hand over their syphilitic cases to syphilis specialists. Salvarsan was replaced by neosalvarsan. During the war supplies of neosalvarsan were not forthcoming, therefore various substitutes were provided, and he presumed they had reached the market through the enterprise of chemists. To pass on one's patients to syphilologists about whose competence one could know but little was not quite to his liking, and it would be useful if ophthalmologists could have better knowledge about the substitutes now being employed. Things were taken on trust, and, instead of getting clearer, the position seemed to be getting more chaotic. If he sent a general syphilitic to the V.D. clinic he received some treatment; but if local treatment was required, for that he made himself responsible. General treatment was continued for a time, and eventually the patient was sent back to him by the V.D. officer to ascertain whether he, the speaker, wished it to be continued. His answer was, that so far as local manifestations were concerned the patient had done well: but he thought it was within that officer's competence to say whether sufficient anti-syphilitic remedies had been given, and he asked whether the Wassermann test had been recently performed. The V.D. officer generally evaded that question, or he said the test could not be regarded as definitely reliable. Therefore the general position was not now more satisfactory than it was before these new drugs had been introduced.

Those who treated this disease still used mercury for the treatment, and they were unable to say absolutely when their cases were cured. The lay public regarded the new method as conclusive and syphilis as definitely curable. If those engaged in treating syphilis would bestir themselves and give more light it would be a great benefit to the whole profession, and the position in this regard would be happier.

3. *Herpes zoster ophthalmicus with internal ophthalmoplegia.*

By M. Baranov.

The following two cases are of interest, as they illustrate an uncommon complication of herpes zoster ophthalmicus.

1. *History.*—Charlotte P—, æt. 68 years. Said to have had erysipelas on L. side of forehead and head in November, 1919. Was ill six weeks. Complained of severe pain over L. eyebrow and temple, for which she attended Moorfields early this year.

Physical signs.—Herpetic scars on L. forehead and adjacent part of scalp. Marked superficial hyperæsthesia and analgesia over first division of fifth cranial nerve.

L. *Cornea*: Clear. *Pupils*: L. larger than R., inactive to light, and no consensual light reaction from R. *Iris*: Disturbance of pigment down and out, otherwise normal. *Lens*: Spot of pigment on anterior lens capsule down and out. *Fundus*: Normal.

V. $\bar{c} - 0{\cdot}5 = \frac{6}{9}$ mostly; $\bar{c} + 5{\cdot}0 =$ J. 1 at 8 in.

R. eye *normal* $\bar{c} + 2{\cdot}0 = \frac{6}{6}$; $\bar{c} + 4{\cdot}5 =$ J. 1.

2. *History.*—Jane C—, æt. 58 years. Said to have erysipelas in March, 1921. Started with pain in R. forehead, followed by an eruption over same area a week later. Two days before this R. eye "went bad," became swollen and closed up. Had no diplopia. Still has pain over R. frontal region.

Physical signs.—Herpetic scars on R. forehead and adjacent part of scalp. Hyperæsthesia and analgesia over the ophthalmic division of the right trigeminal nerve. Almost complete R. ptosis. Ocular movements normal.

Cornea.—R. corneal reflex absent; L. present.

Pupils.—R. pupil dilated and inactive to light and convergence.

Lens.—Peripheral lenticular opacities.

V.—R. = $\frac{6}{36}$; L. $\frac{6}{18}$ partly.

There appears to be little doubt that both these cases of herpes zoster ophthalmicus were complicated with paralysis of the internal ocular muscles. Neither of them had had a mydriatic in their eyes—certainly not since their attendance at hospital. There was slight evidence in Case 1 of a mild iritis, but obviously not sufficient to tie the iris down. It may be mentioned here that ptosis to a greater or lesser extent almost invariably accompanies herpes zoster ophthalmicus with or without involvement of the ocular muscles.

Many cases have now been reported of herpes zoster ophthalmicus complicated by paralysis of one or more ocular muscles, and even by paralysis of the facial nerve.

A review of the condition with its complications is given by Burton Chance (1).

Traquair (2) gives a short *résumé* of cases up to date.

Hutchinson (3) reviews the condition, which at that time was beginning to interest ophthalmologists, and gives a list of cases.

The following cases have been singled out as being similar in nature to the two cases given above:

(*a*) *Herpes with dilated pupil* (4).—Considered to be due to impairment of third nerve. It is of interest that in this report he quotes a case of Broadbent's where there was herpes affecting the arm followed by muscular impairment.

(*b*) *Hutchinson* (5).—Complete third nerve palsy. Complete recovery.

(*c*) *Herpes zoster ophthalmicus with ophthalmoplegia totalis* (6).—All ocular movements recovered with the exception of the sphincter.

(*d*) *Mydriasis following herpes* (7).

In all these cases therefore we have, in addition to the sensory loss, an involvement of certain motor fibres. It is possible that the acute inflammatory process, which apparently involved the Gasserian ganglion, spread and involved the

ocular motor nerve in the cavernous sinus. One would have expected, however, if such were the case, more motor loss. In the two cases quoted above the internal ocular muscles were alone affected, and it seems probable that the lesion here was in the ciliary ganglion, where the cell stations of the fibres of the third nerve supplying these muscles lie. Herpes zoster ophthalmicus is very probably an acute specific infection, and though the virus appears to have a special predilection for sensory ganglia, it seems possible that occasionally other parts of the nervous system are affected.

Galezowski and Beauvois (8), after giving examples of two cases of herpes with sixth nerve palsy, proceed to review the subject, and their opinion of the pathology (on clinical grounds) is that it is a neuritis of the fifth nerve, with inflammation spreading along the anastomotic twigs (described by Sappey) between the first division of the fifth nerve and the motor nerves of the eye.

On the other hand, they quote Sulzer, who considers the muscular palsies as evidence of an encephalitis.

Finally, Willbrand and Saenger (9) incline to the view, in spite of the work of Head and Campbell, that herpes is a neuritis, though in certain cases an affection of the spinal ganglion may be found as well. They consider this particularly so as herpes is so often combined with motor palsies. A neuritic or perineuritic process may involve such nerves as are in anatomical contact, as in the cavernous sinus. The combination of occipital herpes with facial palsy can only be explained by the virus affecting more nerves than one, or that the neuritis spreads by direct continuity through anastomoses.

I have to thank Dr. Gordon Holmes, and Messrs. J. H. Fisher and Malcolm L. Hepburn for allowing me to show their cases.

References.

(1) *Annals of Ophthalmology*, 1915, xxiv, 693.

(2) *Ophthalmic Review*, 1913, xxxii, 65.

(3) *Royal Ophthalmic Hospital Reports*, London, 1866-67, v and vi.

(4) *Ibid.*, Hutchinson, 1866, v, 209.
(5) *Ibid.*, Hutchinson, 1867, vi, 183.
(6) *Annals of Ophthalmology*, 1907, xvi, 120.
(7) *Lyon Méd.*, 1920, v, 458.
(8) *Recueil d'Ophtalmologie*, November, 1906, 660.
(9) *Neurologie des Auges*, 1901, ii, 202.

Mr. Stack asked for information as to the anæsthesia of the cornea which sometimes occurred in these cases. One old lady patient of his had an anæsthetic cornea after herpes ophthalmicus: for two years she had absolute anæsthesia of the cornea. By putting in parolein every night she avoided ulceration of the cornea. She also had some conjunctivitis. It was the only case he had met with in which the condition continued such a long time.

Mr. W. T. Holmes Spicer said it was not uncommon to find anæsthesia lasting a long time after herpes ophthalmicus. He had known it to last five years in one case. A man had herpes and very severe irido-cyclitis. The eyeball shrank, and had to be removed, and the operation was done without an anæsthetic. He only felt pain for a moment, when the scissors were put down behind the globe; this must have stimulated some nerves which did not come from the Gasserian ganglion.

Dr. Gordon Holmes said the three cases were referred to him by his surgical colleagues at Moorfields. He had not yet been able to glean from the literature what was regarded as the pathology of the condition. An important point in the pathology of herpes which had been lost sight of in this country, chiefly owing to the form in which Dr. Henry Head and Dr. Campbell put forward their researches on this disease, was the extraordinary diffuseness of the lesions. For instance, even though there might be a herpetic eruption limited to the 8th dorsal segment, severe changes were generally found in the neighbouring ganglia too. The late Dr. Alexander Bruce, of Edinburgh, showed that sometimes the central nervous system might be also involved. In the present cases the chief incidence of the disease certainly fell

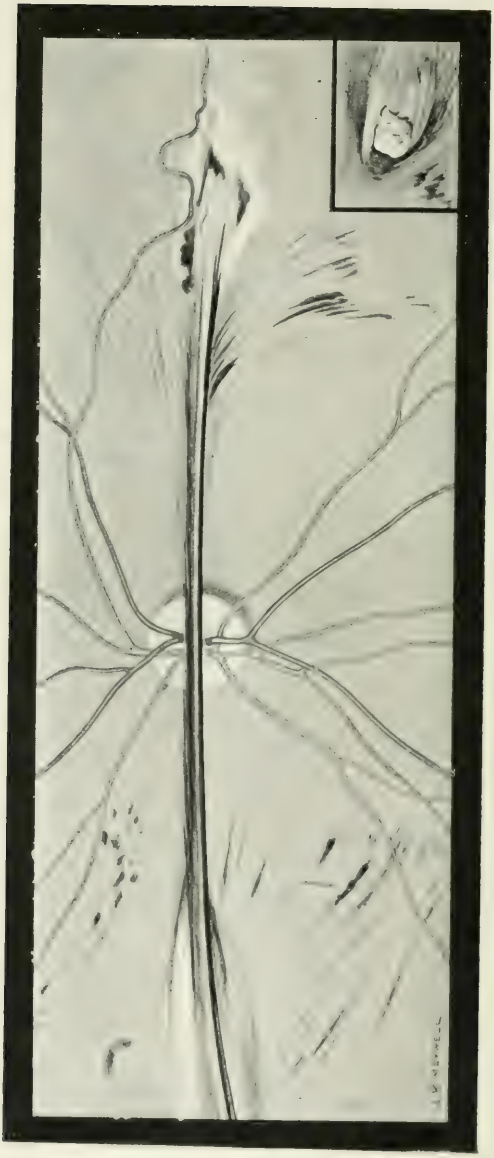

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PLATE V.

Illustrates Mr. J. F. Cunningham's paper on A Case of Intra-ocular Foreign Body (p. 171).

on part of the Gasserian ganglion, but there must have been also lesions elsewhere. He was not in a position to suggest the exact sites of the lesions which caused the ocular symptoms in these cases, but they are most probably due to affections of the ciliary ganglion.

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4. *A case of intra-ocular foreign body.*

By J. F. Cunningham.

(With Plate V.)

Pte. A- , wounded in France by a splinter from a machine-gun bullet October 18th, 1916. Seen at the Boulogne base soon afterwards. I am indebted to Sir William Lister for the loan of the drawings shown with the case, which were made by Sergt. Maxwell, and for the field, which was taken by Sir William Lister himself.

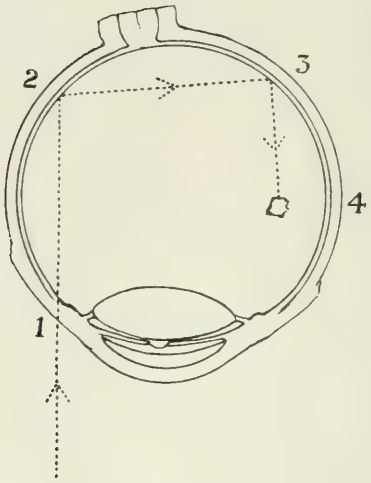
R. V. $\frac{6}{6}$. L. V. was then $\frac{6}{60}$.

In the left eye, on the inner side, about on a level with the centre of the cornea, is a depressed scar in the sclera. On ophthalmoscopic examination there is a stout cord-like band passing back from the position of the scar to the retina on the inner side, where there is a white area with considerable pigmentation surrounding it. This area is roughly about twice as large as the disc. There is a similar area on the temporal side of the disc, and between them a broad flat sheet formerly passed, obscuring the view of the centre of the O.D. When the eye looked straight forward there was a glistening foreign body about 5 D. forward in the vitreous, and a little below the last area described on the temporal side.

The F.B. was not moved by the Haab magnet, and the patient felt no pain when it was in action. The presence of a F.B. was, to the best of my recollection, also confirmed by X rays.

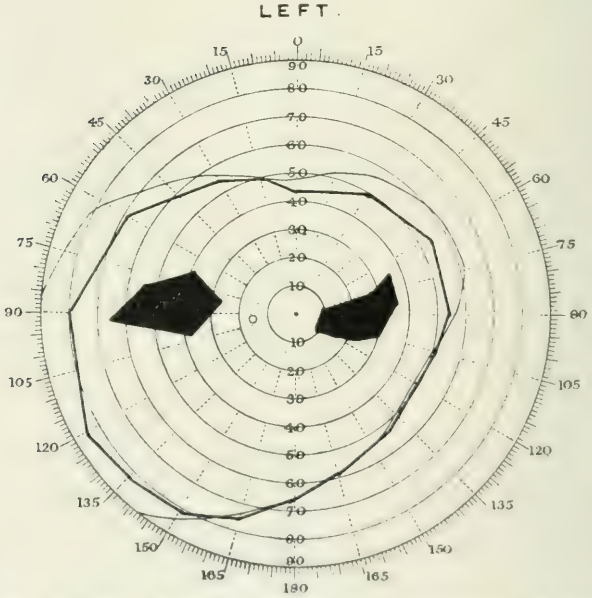
The eye at the present time is quite quiet, though vision is now reduced to finger-counting. The most striking change in the fundus now, *i. e.* after a lapse of over $5\frac{1}{2}$ years, is that the broad black sheet, passing between the two areas of disturbance in the fundus which are the first and second

Fig. 3.



Course of the F B. in the globe.

Fig. 4.



Pte. A—, 12th Suffolks. May 24th, 1917. 10 mm. sq White. Good light.

points of impact, has been replaced by some fine lines resembling telegraph wires seen from some distance. The change is most striking if it is compared with the drawing made in 1916 (Pl. V). The F.B., which is practically in the same position, is surrounded by a certain amount of fibrous tissue. In order to produce two ricochets, and to come to rest in its present position, the F.B. must have perforated the globe with considerable velocity.

I saw this patient once, a year or two ago, and I noted that the black sheet had disappeared then.

Mr. Harford, who saw him at a Pension Board recently, kindly brought the case to my notice.

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5. *Retraction of the upper lid, with defective upward movement of the right eye.*

By J. F. Cunningham.

W. T—, male, æt. 14 years. The condition has been present since birth as far as can be ascertained.

R.V. $\frac{6}{18}$. Fundi normal. L.V. $\frac{6}{6}$.

He has been under treatment for trachoma at the Ophthalmia Isolation Schools, Chigwell. The lids are now quite smooth, and, except for slight discharge the first thing in the morning, he is free of the disease. The condition is not due to this disease. The pupils are equal and there is no proptosis. The left upper lid is in the normal position. In the right eye the sclera is exposed above the cornea when he looks straight to the front. On attempting to look up the movement of the right eye is defective. Mr. Lawford pointed out that there was also some retraction of the skin below the right lower lid when he attempts to look upwards.

Mr. J. B. Story said he was very glad to have had the opportunity of seeing the first of these two cases, as last year he had the case of a small boy who got hurt in the lower lid of the left eye from the explosion of a percussion cap which a boy had when playing with him. There was a small linear scar 2 or 3 mm. in length on the outer surface of the

lower lid, close to the inner canthus, and at first he took it to be a mere scratch on the skin. But on looking on the inside of the lid he saw a similar cut. A sharp splinter of metal had gone through the lower lid and passed into the conjunctiva. There was no disagreeable feeling in the fornix, and apparently nothing was there. There was a scar on the eyeball corresponding to the position of the cut on the inner surface of the lid. The only fault the boy found with his eye was that there was a dark spot, and a scotoma was found in the place corresponding to where the foreign body had entered the eyeball; the scotoma was upwards and outwards. After close examination with the ophthalmoscope, taking much trouble about it, he was only able to detect at the extreme periphery, inside the retina, that there was a whiteness—some apparent œdema of the retina; it tallied with appearances following concussion. After two or three days this white membrane went back a little, and he could see hæmorrhages and choroidal pigment scattered about the retina at the extreme periphery. This also was a non-magnetic body: it was a white metal case, and Haab's magnet had no influence upon it. He was unable to derive any very certain help from the skiagram, and the skiagrapher could not be certain whether the body was inside or outside the eyeball. Therefore the speaker thought it better to leave it alone. If it had been definitely found to be on the surface of the sclerotic it might have been removable. The boy had been all right since: his vision was $\frac{6}{6}$, but he still had the scotoma, and the field of vision was not now so large as it was at first. From what he now saw of Mr. Cunningham's case he felt satisfied it was the best policy to leave the case alone. Much experience was gained on this subject during the war.

Mr. M. S. Mayou, discussing the first of the cases, said that some years ago he had a boy who was out shooting, and a pellet struck him in the eye. Nothing was done: the eye quieted down, and the foreign body could be seen sticking in the fundus near the disc. Four or five years later the boy was going through a gap in a hedge and fell into the ditch, and three days after he came up with intense iritis –

almost suppurating. He removed the eye, and found that the foreign body had fallen out from its original position in the fundus to the bottom of the globe, evidently as a result of the fall into the ditch. Why the eye should have become so inflamed in consequence he could not tell. Possibly organisms might have been carried in at the time the foreign body entered, or there might have been a subsequent metastatic infection. Perhaps some who had seen war injuries could throw some light on it.

Mr. A. L. WHITEHEAD said he did not think a ricochet after the entry of a foreign body into the eye was common. He had only seen one such case, and that was a short time ago. The man was working at a steel-cutting machine when a small fragment came off and entered the eyeball at the corneo-scleral junction on the outer side. A small hole could be seen at the periphery of the iris. The lens was uninjured. The foreign body had passed through the suspensory ligament of the lens, and could be seen stuck in the retina on the inner side. He found a small tear in the choroid and retina, with a hæmorrhage over it, on the outer side, therefore the fragment must have ricochetted across. The present case was a much more striking one.

Mr. J. B. STORY, in further comment, said he had seen a number of injuries from pellets and shot, but he had not yet seen a case where a grain of shot entered the eyeball or passed through it in which the eye could be saved; every eye had to be removed. Possibly that was because of the septic nature of the pellets and shot used in Ireland, producing sepsis and an inflammatory reaction.

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6. *A series showing the results of West's operation (per-nasal dacryocystostomy).*

By C. I. GRAHAM.

MR. GRAHAM said he had described the operation to members. But there was one point he wished to direct attention to now, namely, the introduction of the wire in establishing the fistula. This he regarded as the most important part of

the operation, and was originally suggested by Mr. Leslie Paton. The speaker had done it in every case since that suggestion was made. The cases seen to-day, some of which were operated upon in 1914 and 1915, had the operation performed in that manner. Since the eighth day after the operation he had not seen those patients until he wrote for them to come up; therefore they were not selected cases. They showed the absolute result of the operation as he had described it.

Mr. Holmes Spicer said the members felt very grateful to Mr. Graham, because diseases of the lacrymal apparatus were never entirely welcome to the ophthalmic surgeon. Treatment was not very satisfactory. Any method of cure would be welcomed, even though it might not be carried out by ophthalmic surgeons.

The President said members thanked Mr. Graham very much for showing such excellent results. He, the speaker, had asked rhinologists to perform this operation for mucocele, but he had not generally seen anything like such a satisfactory result, and, apparently, without any risk of failure. He understood Mr. Graham attached great importance to the introduction of the wire. He believed there had been successes by other rhinologists by operation, but they appeared to have been of a temporary character. The results of these operations, however, appeared to be permanent.

Mr. J. B. Lawford asked whether the operation had been performed during the acute stage of the condition. Did Mr. Graham undertake it when there was pus in the lacrymal sac?

Mr. Leslie Paton said he had never regretted sending a case of this kind to Mr. Graham, but he never sent them to him in an acute condition. That was done once, and it was not altogether a success. An attempt was first made to get the sac free of pus and discharge; they simply had some mucous discharge when they were sent to be operated on.

Mr. Traquair said that in the Edinburgh Infirmary they had sent many cases of this nature to Dr. Fraser, who had done the operation with success in the presence of abscess or

fistula. His results were published in the *British Journal of Ophthalmology* in 1919, and were very satisfactory.

Mr. J. B. Story (Dublin) said he had been delighted to see this series of cases to-day. For some years, indeed since the publication of West's original description of the operation, they in Dublin had been having these cases of lacrymal sac disease operated upon by Mr. T. O. Graham (a namesake of the operator on the series now shown), and no attempt was made to get the sac less septic before asking Mr. T. O. Graham to deal with them. That gentlemen had operated upon a great number of sacs successfully, many of which were secreting pus and mucus at the time of the operation. He, like Mr. Paton, had had no cause to regret sending these cases for operation, and if Mr. T. O. Graham's results were seen by the members, they would have as much confidence in him as many now had in Mr. C. I. Graham. If he, the speaker, had an abscess in his lacrymal sac, he would prefer to have it excised than to have a West operation performed, because he thought the latter was a disagreeable one for the patient, notwithstanding what he had said as to the success of the operation. He had been very glad to see the beautiful examples of what the operation could do.

Mr. Graham, in reply, said he had not had the opportunity of doing this operation in the acute stage. In reply to Mr. Story, he did not know of patients complaining of any discomfort or unpleasantness, therefore he did not think that could be put forward as a contra-indication for it, *i. e.* for abstaining from doing the operation.

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7. *Two cases of tuberculous iridocyclitis. Clinical notes and pathological report, with illustrations.*

By Humphrey Neame.

Read by F. A. Williamson-Noble.

(With Plate VI.)

Case 1 has already been described in detail, namely, at a meeting of the Ophthalmic Section of the Royal Society of

Medicine, and therefore will be disposed of briefly. The paper dealing with this case has been accepted for publication in the *British Journal of Ophthalmology*, 1922, vol. vi, p. 204.

Herbert D—, æt. 19 years, was admitted to the Royal London Ophthalmic Hospital under Mr. E. Treacher Collins's care in June, 1921, and underwent the operation of excision of the left eye on account of its condition. There was advanced tuberculous iridocyclitis of this eye, with staphyloma of the opaque cornea.

A lantern-slide exhibited showed the macroscopic appearance of the eye divided in the sagittal plane, with the slight staphyloma and infiltration of the cornea, almost complete obliteration of the anterior chamber with a mass which is partly continuous with cornea in front, with iris and ciliary body on each side. The slide was from one of a pair of stereoscopic photographs, and was passed round for inspection. The specimen itself was mounted in a bottle.

A microphotograph was shown of a section of part of the mass which occupies the anterior chamber, including portion of the cornea, and posteriorly some of the uveal pigment tissue. The section showed the presence of blood-vessels in a fibro-cellular mass, and one giant-cell of Langhan's type near its posterior limit.

A microphotograph showed the cornea near its centre. The epithelium and Bowman's membrane above, below a cleft with sinuous margins—separating Descemet's membrane from the deep corneal layers. In the very middle of the thickness of the cornea were visible several blood-vessels, containing red blood-corpuscles enclosed within a well-defined endothelial layer. The deeper layers of the cornea were infiltrated with cells. Posterior to Descemet's membrane was a small portion of the mass which filled the anterior chamber.

The tuberculous affection was limited in this eye to the anterior part of the uveal tract—the iris and ciliary body—and the immediate extension therefrom into the cornea.

In addition to the lesion in this eye, there was a chronic

fibrosing form of tuberculous lymphadenitis of the glands of the neck, and also tuberculosis in the form of small nodules in the palpebral conjunctiva of the other eye.

A further microphotograph was shown of two adjacent nodules in the conjunctiva. It showed an incomplete conjunctival epithelium above, vascular subconjunctival connective tissue below, and two aggregations of lymphocytes, in one of which was a fairly well-defined giant-cell lying among epithelioid cells.

This patient was seen in February this year, and remains in fair general health. There were still some minute nodules in

FIG. 5.

CASE 2.—Tuberculous iritis.

the conjunctiva of the right eye, but the glands of the neck had shrunk to a very small size.

CASE 2.—The second case is of particular interest, since the late after-history is known.

Alice A—, at the age of four years, was admitted to the London Hospital under the care of Mr. A. Bruce Roxburgh, on July 31st, 1907 (No. 1766/1907). The clinical notes that follow are copied from the case-notes made by Mr. D. Davidson, who was House-Surgeon to the Ophthalmic Department at that time:

July 31st, 1907: Fell off a chair *eight months* ago and was unconscious two hours. Hæmorrhage seen across the right eye after. *Four months* ago right eye began to look strange. Attended O.P.'s April 24th, 1907, when the cornea was described (see 1067/1907) as hazy, stippled. K.P. + +. Iris covered with nodules of vascularised lymph. Admission

May 8th, 1907. Had two injections of tuberculin (no notes of opsonic index).

Developed measles and was discharged July 1st, 1907.

Readmitted July 30th, 1907.

Left eye clear. Lungs clear. Heart clear. Glands, none felt.

Right eye: Some drooping of upper lid. Slight conjuncti vitis and circumcorneal congestion. The lower part of cornea is hazy and covered with fine stippling.

Nodular masses of yellowish-grey colour sticking up from iris.

Pupil irregular and almost immobile. No view of fundus obtainable.

August 1st, 1907: Operation by Mr. A. B. Roxburgh. Excision right eyeball: ball sewn into Tenon's capsule.

Fig. 5 has been drawn from a diagram in the House-Surgeon's notes, and shows the extent of involvement of the iris.

A similar diagram made by the previous House-Surgeon—Dr. W. B. Billinghurst—at the time when the child was first admitted to the London Hospital, shows that very slight alteration had taken place during the interval of ten weeks.

Pl. VI, fig. 2, is a pair of stereo-photographs of the museum specimen. Both the stereo-photographs and the specimen are exhibited. These show the cross-section of a nodule near the pupillary margin of the iris on one side, which nodule is less than 0·5 mm. in diameter, and two nodules in the ciliary body on the same side, each of which is about 1 mm. in longest diameter. There is also a more diffuse white infiltration of the posterior layers of the iris near its root.

Pl. VI, fig. 1, is a microphotograph of the anterior part of the eye on one side. In it are seen a mass on the front of the iris, and fibrous bands connecting this with the posterior surface of the cornea: closure of the angle in the extreme periphery: and a larger conglomerate mass of nodules involving the ciliary processes and root of iris. The minute structure of these nodules is that of a fibrous cellular tissue. There is no evidence of caseation.

A photograph showed the patient as she was in March, 1922, at the age of 19 years—fifteen years after excision of the

PLATE VI.

Illustrates Mr. Humphrey Neame's paper on Two Cases of Tuberculous iridocyclitis (p. 177).

Fig. 1.—Case 2: Tuberculous iridocyclitis. Microphotograph showing nodular infiltration of iris and ciliary body.

Fig. 2.—Stereo-photograph of museum specimen. Shows a white nodule in the iris at pupil margin, and a mass in the ciliary body.

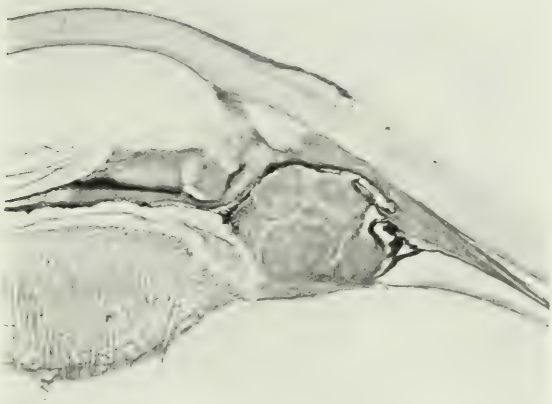

Fig. 1.

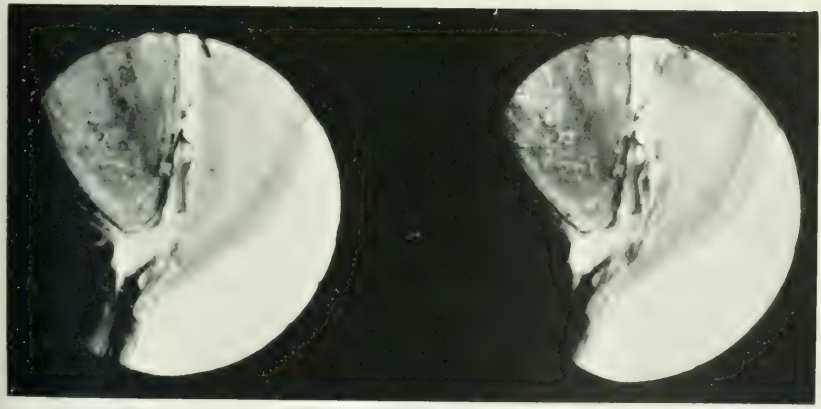

Fig. 2.

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tuberculous eyeball. She and her mother both stated that she was in the best of health, was at regular work, and was able to take as much exercise as any of her friends, and that she was very fond of dancing, in which she indulged to a great extent without inconvenience.

This may fairly be taken as a case of primary tuberculous iridocyclitis in which complete recovery followed the excision of the diseased organ.

The writer acknowledges his indebtedness to Mr. Roxburgh and to Mr. Collins for the use of the notes, and to the reader for so kindly taking his place in his unavoidable absence.

8. *An extra-dural tumour of the optic nerve.*

By M. L. Hine.

(With Plates VII and VIII.)

In the *Royal London Ophthalmic Hospital Reports* for 1912 A. C. Hudson published an exhaustive paper on all cases of optic nerve tumour recorded up to that date, amplifying Sir John Parsons' previous work on the subject. In all he collected 182 cases, and classified them into three main groups, leaving out some 29 cases which could not well be included in any group. These groups were as under:

A. Gliomatosis of the nerve, of which he found 118 examples.

B. Fibromatosis of the nerve-sheath, of which there were only 6 cases.

C. Endothelial tumours of the nerve-sheath, of which there were 29 cases.

Of the six cases recorded in group B, four occurred in children under 7 years of age, one in a woman aged 21, and another in a woman aged 31, though in this last case exophthalmos had been present for 20 years. Only one of the cases occurred in a male. The main points of clinical interest in this type of case are given as—

1. The slow rate of increase.

2. The presence of exophthalmos prior to the onset of defective vision.
3. The absence of recurrent growth after incomplete removal, as in Sir John Parsons' case reported in 1903, suggesting their innocent character.

Since this paper was published a few additional cases of optic nerve tumour have been reported, but I have only found records of seven. Of these six were intra-dural and one only extra-dural. This was reported by Sattler in 1913 as a "fibro-sarcoma of the optic nerve-sheath." The age of the patient is not stated. The tumour was of slow growth, and vision was not affected until five months before operation. The nerve was running intact through the tumour, which was composed of rather dense fibrous tissue infiltrated with small round-cells in variable numbers. In places these cells were found in clumps. Here and there through the tumour were found cells of an epithelial type with a large oval nucleus, and also a few cells with a spindle-shaped nucleus. The tumour had a few blood-vessels, but was comparatively non-vascular.

The case I am reporting to-day presents many points of interest, which can best be indicated after giving you the clinical history and pathological report.

Henry H—, æt. 69 years, a painter by occupation, attended at the Royal Westminster Ophthalmic Hospital on June 8th, 1921, for the purpose of getting some glasses for close work. A little watery homatropine was instilled into each eye, for complete examination, as his vision was not quite satisfactory. Beyond a few lens striæ in either eye and general arterio-sclerosis of the retinal vessels nothing abnormal was noted.

R. V. $\bar{c}$ — 0·5 D. sph. = $\frac{6}{12}$.

L. V. $\bar{c}$ — 1·0 D. sph. = $\frac{6}{9}$.

On the 22nd inst. he returned, complaining that he had been unable to see with his right eye for a week. The right pupil was active. Tension was normal. Vision—hand-movements.

There was a marked "choked disc" in that eye, with

several hæmorrhages into the surrounding retina. The amount of swelling was not noted at the time, but the following week, when the hæmorrhages were much more extensive, it was noted as being 4·0 D.

There was no similar affection of the left O.D.

There was marked general arterio-sclerosis, the temporal arteries standing out like cords. The urine contained no albumen or sugar, and its sp. gr. was 1014. The Wassermann reaction was not done at that time, but more recently it has been done, with negative result.

Dr. Gordon Holmes kindly saw him for me at Charing Cross Hospital, and reported that there was no disease of the nervous system. The heart was very much hypertrophied and the blood-pressure very high. On auscultation over the right temple a continuous hum could be heard, suggesting the possibility of an intra-cranial aneurysm.

For two months he was taking pot. iod. gr. x *t.d.s.* from Charing Cross Hospital, reporting at intervals for observation of his fundus. The swelling of the disc gradually subsided, until on October 10th I noted "now secondary optic atrophy, right." There was at this time P.L. only.

Towards the end of August, some two months after the onset of the defective vision and papillitis, he began to complain of severe pains and headache in the right temple, and over the right side of the forehead. At the end of October the right eye showed some injection of the conjunctiva of the lower fornix, and slight proptosis.

The injection of the conjunctiva increased, and some chemosis was noted. Early in December Mr. Waggett, who kindly saw him for me at Charing Cross Hospital, recommended an exploratory puncture of the sphenoidal sinus, a skiagram showing a shadow, suggesting pus, posterior to the right orbit. Exploratory puncture of sphenoidal and other sinuses proved negative.

After this the swelling of the conjunctiva of the lower fornix increased considerably, the proptosis also increased, the pain in the right temple grew more and more severe, and the eye became totally blind. There was no fulness of the retinal veins.

I did not note the range of ocular movement. The proptosis was directly forwards.

He was taken into Hospital on February 8th and on February 11th was given an anæsthetic, full permission having been obtained to do anything that was thought advisable. The lower fornix was by this time obliterated, the conjunctiva bulging between the lids. This bulging conjunctiva was incised and the orbit explored with a director. A tense elastic mass was found to lie directly behind the globe. This was punctured to eliminate the presence of pus, but proved to be solid.

The globe was then excised, and the whole orbit, within the cone of muscles behind it, was found to be filled with a solid mass, with a concave anterior surface corresponding to the convexity of the globe. The optic nerve had been cut flush with the globe and the proximal cut end could be seen definitely embedded in the mass.

By working about with one's finger and a pair of curved scissors with closed blades it was found possible to isolate the orbital mass on all sides, and, finally, to tear away the whole mass from the apex of the orbit. Part of the inferior rectus was found attached to the growth after removal, otherwise it appeared to have been cleanly separated from the surrounding structures.

For the following pathological report I am indebted to Dr. F. Taylor, Pathologist to the Royal Westminster Ophthalmic Hospital :

" The specimen consists of a right eye with a mass of tissue posteriorly consisting of the orbital contents within the cone of muscles. A portion of the inferior rectus has been torn away with the mass, and remains attached to it at its posterior and lower part.

" The mass is irregularly pyramidal in shape, extending from immediately behind the globe to the apex of the orbit. Anteriorly it closely ensheathes the posterior part of the globe, being hollowed out to form a sort of false socket for the latter, with which it does not appear to have been intimately fused. The apex of the growth was situated at the optic foramen, where it was seen to closely

PLATE VII.

Illustrates Mr. M. L. Hine's paper on An Extra-dural Tumour of the Optic Nerve (p. 181).

Fig. 1.—Vertical longitudinal section of globe and tumour. The gap between the globe and the tumour is artificial, the globe having been removed separately.

Fig. 2.—Vertical transverse section of anterior portion of bisected tumour, showing portion of nerve and infiltrated nerve-sheath and sub-dural space, and tumour tissue (low power).

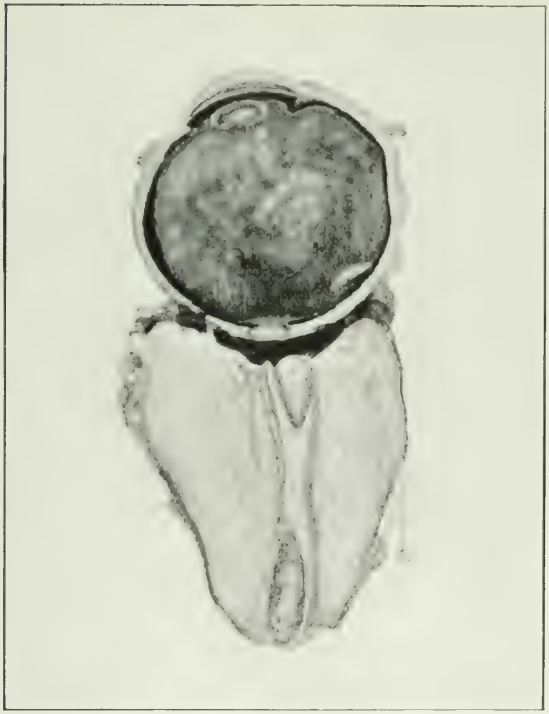

Fig. 1.

Fig. 2.

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PLATE VIII.

Illustrates Mr. M. L. Hine's paper on An Extra-dural Tumour of the Optic Nerve (p. 181).

Fig. 3.—Section of tumour showing extreme arterio-sclerosis of vessels.

Fig. 4.—Section of tumour showing cellular infiltration.

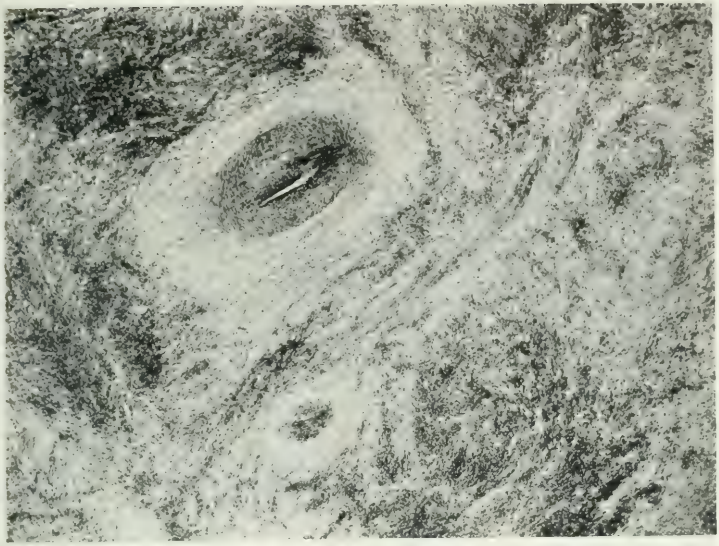

Fig. 3.

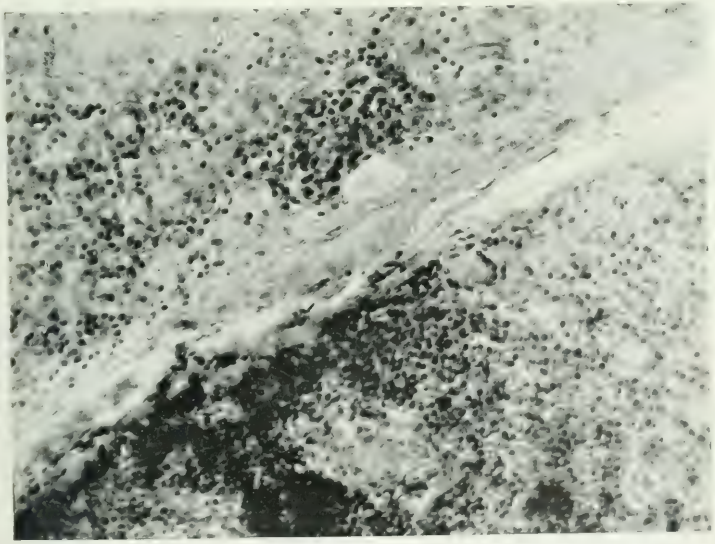

Fig. 4.

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surround the optic nerve, and does not seem to have extended through the optic foramen.

"On macroscopic section the mass measures 28 mm. from cut distal end of optic nerve to the apex. Its greatest length is 32 mm. The greatest vertical diameter is 27 mm. and its breadth 26 mm. It consists of dense white fibrous tissue of uniform consistence, through which the optic nerve runs apparently intact, the tumour tissue being evidently continuous with its dural sheath. The cut end of the nerve is surrounded by the growth. The outer surface is rough and shaggy, from the presence of numerous tags of surrounding orbital fat and cellular tissues, and part of the inferior rectus, as mentioned above, is adherent to its lower surface. No gross changes are seen in the eye.

"On microscopic examination the tissue of the tumour consists of cellular tissue, containing fibroblasts, and infiltrated with small round-cells, this infiltration being very variable in amount in different parts of the section, in parts forming dense masses, and in other parts being very diffuse. The cells constituting this infiltration comprise small lymphocytes and plasma-cells, with an occasional eosinophile. The cellular tissue itself in parts shows some hyaline degeneration. The nerve-fibres appear normal, but there is some infiltration of the interstitial tissue, and similar infiltration is present in the sheaths of the nerve and subdural space.

"The blood-vessels form a prominent feature of the section. They are abundantly present, and of various sizes, and all show extreme thickening of the media and intima, and endothelial proliferation, producing, in some places, endarteritis obliterans which quite closes the lumen of the vessels. There is some softening of the greatly thickened media of the large vessels.

"There is an absence of giant-cell systems and of endothelial cells in the masses of cellular infiltration.

"These histological appearances suggest that the tumour is a chronic inflammatory mass, the absence of giant-cells making tuberculosis unlikely, and the presence of changes in the vessels suggesting a syphilitic origin. The Wassermann reaction is, however, negative."

While, in general appearance, this growth certainly seems to resemble the rare examples of extra-dural tumour of the optic nerve grouped under the heading of "fibromatosis," there are several points in the clinical history which do not correspond with that of previously reported cases. These are—

(1) The age of the patient, viz. 69 years, as previously the oldest recorded case in this group was 31, and in this patient the eye had been proptosed for twenty years.

(2) The onset of defective vision as the first symptom, the proptosis not coming on until some four months later.

(3) The comparatively rapid increase of the "tumour," as evidenced by the increasing proptosis over a period of four months.

Mr. M. S. Mayou said the appearance of the photographs suggested a chronic inflammatory condition, but he understood the Wassermann was negative.

Mr. Frank Juler said he had at Moorfields a few years ago a case of proptosis in an old man, which came on in a way somewhat similar to that in Mr. Hine's case. He had previously had an operation done for glaucoma, but that had nothing to do with the condition for which he attended. There was proptosis of two or three months' duration, and finally ulceration of the cornea, and it was necessary to remove the eye. Even then he was not sure there was growth in the orbit: if there was, it was a very soft elastic mass. Later the man began to get proptosis of similar nature in the other eye, and he exenterated the orbit on the affected side. The orbital tissues presented microscopical appearances somewhat similar to those in Mr. Hine's case, except that the mass was not localised as a definite tumour, but had infiltrated the orbital tissues. There were, here and there, infiltrations of inflammatory cells. After the exenteration of the orbit, the patient had some lung trouble, and died in hospital. It was not possible to secure a *post-mortem* examination to see whether there was extension from the brain, or whether there was a similar condition on the other side.

But he examined the exenterated orbit, and there seemed to be a small perforation on the inner side of the orbit, suggesting a communication with the nasal sinuses posteriorly. The pathologist thought it was of the nature of a fibromatosis. The condition coming on in the second eye favoured the dea that it was merely inflammatory, and that in some of these cases there was an inflammatory basis, with a proptosis simulating tumour.

Mr. Roper said he was able to recall a similar case. It was in an elderly lady, *i. e.* 84 years old, now living and aged 92, and when he called to see her she had what appeared to be orbital cellulitis. He advised certain treatment, and left. A fortnight later he was sent for. She was then in great pain, the swelling had increased, and he concluded it was suppuration, not ordinary orbital cellulitis, but due to a growth. He was unable to examine the fundus, as the lens was opaque. He first excised the eye, then exenterated the orbit, taking out what appeared to be a sarcoma. But on examining the eye the pathologist said it was a granuloma—a chronic inflammatory condition. There was no recurrence in the orbit, but a somewhat similar condition occurred a few months later on the other side, with considerable proptosis. This subsided. He did not see the case again, but the medical attendant said he thought the other eye was going the same way as the first had done, but it had then subsided, and sight had been retained. Both the cases related to-day were in feeble people, and the question arose whether in such people the severe operation of exenteration should be done. This lady, however, survived, and she was relieved of her intense pain. The man mentioned in the paper was only 69, but he had arterial sclerosis and other signs of old age. He was informed that the vision of the other eye had been lost since, but did not know the cause.

Mr. Johnson Taylor remembered a case of this nature in a woman aged 30. This patient had gradually increasing proptosis and some of the signs of orbital cellulitis. He put her upon large doses of iodide of potassium and mercury, and pushed the treatment, but that had no effect on the

condition. He therefore concluded it must be something of a malignant nature. He excised it, and exenterated the orbital contents. When it was examined afterwards, the pathologist said it was an organising gumma. Some little time after the same condition appeared in the fellow eye, which also became proptosed. Armed, however, with the knowledge about the other eye, he pushed the mercury, so much so that the patient had dysentery; and after that state had been reached the eye condition cleared up.

The PRESIDENT considered there was some value in Mr. Johnson Taylor's remarks. He, the speaker, had felt for some time past, especially since he had been handing over syphilitic cases to colleagues, that those who met these cases were apt to be only half-hearted in the administration of well-known anti-syphilitic measures. People persisted in giving only 10 gr. of iodide of potassium three times a day, trusting to that to produce results. That was not the kind of food that he was brought up on by a general surgeon, who was an efficient syphilologist. He gave 20- to 30-gr. doses of iodide of potassium, combined with the administration of mercury, and he got with that treatment results when others failed.

The cases referred to by Mr. Hine, Mr. Juler, Mr. Roper and Mr. Johnson Taylor formed an interesting group, and some of them belonged to a group different from those of which Mr. Hine's was an example.

Mr. HINE, in reply, said he thought, when he took the eye out, there was a malignant growth in the orbit. When he asked that the title might be put down as "unusual tumour," it had been suggested it might be a gumma. He therefore took the specimen and slides round to show certain people, including Mr. Greeves, Sir John Parsons, and Mr. Hudson. Mr. Greeves and Sir John Parsons said they thought it would be quite safe to put it into the fibromatosis group, in spite of the fact that the clinical history did not quite fit in. Sir John Parsons thought it was very much like a twin-sister of a case he described in 1903. It had been said that nobody knew much about these tumours. There was

work still to be done on such tumours, which were rare, and on tumours of the optic nerve in general. He regretted the absence of pathologists, whose opinions he wanted to hear, though he had been interested to hear of the case mentioned by Mr. Juler, which was evidently inflammatory in origin and not so definite a tumour as in the present case.

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9. *Recurrence of endothelioma of orbit after twenty years.*

By W. T. Holmes Spicer.

The history of this case goes back to 1902 when the man had a tumour between the globe and the upper margin of the orbit. That was removed. Four years later a recurrence, and the eyeball was removed. Five years later still the orbit was exenterated. The man now came with a fourth recurrence: the growth had been very slow, but there was a definite change during the last two years. Previous examinations had shown that the tumour was an endothelioma.

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10. *Intra-ocular cholesterin tumours.*

By F. A. Williamson-Noble.

(With Plates IX and X.)

The question of diagnosis of intra-ocular tumour is frequently a difficult matter, and the diagnosis may still be difficult even when the eye has been excised and submitted to pathological investigation. One source of error is that masses of cholesterin resulting from a hæmorrhagic lesion may mimic a tumour very closely. As I have recently had a case in which this occurred, and through the kindness of Mr. Mayou have had access to two other cases, I felt it was worth while describing them to you. I propose to describe one of Mr. Mayou's first.

The first case, then, occurred in a man who had had an injury to the eye five years previous to excision. There was slight

conjunctival injection: the tension was slightly lowered. The iris bound down and *bombé* above. The lower half of the A.C. was filled with a whitish mass of glistening character fringed with blood-pigment at its lower margin and sharply defined above. The lens is absorbed and the retina detached.

There is also an organised choroidal exudate at the posterior pole of the globe, showing calcareous changes. Unfortunately no sections were made of the specimen, as the other half of the eye was sent to the College of Surgeons' Museum, but from examination with a loupe one would have no doubt that the mass consisted of lamellæ of fibrous tissue enclosing spaces filled with cholesterin.

The second case is an eye sent to me for pathological examination by Dr. Bardsley. The history is as follows:

Mrs. G—, æt. 48 years, seen on January 14th, 1922. Confinement two years ago, and sight of L.E. became dim shortly after the birth of the child: her sight became steadily worse until the eye became blind. January 1st: Lids, eye and surrounding cheek became swollen with intense pain in the eye. Condition when seen: Intense chemosis; pupil widely dilated and inactive to light. T. 2; no P.L. Scarcely any red reflex, but a large mass could be seen coming far forward into the vitreous. Iris covered with large new vessels; on microscopic examination the pupil was seen to be vertically oval; there were bluish swellings in the regions of insertion of the superior, inferior, and internal recti, which on section could be seen to be occupied by blood-clot. There is a fine membranous formation in the vitreous, and at the back of the eye is what appears to be a small growth showing some pigmentation.

Pl. IX, fig. 1: The appearances are shown in this figure, where one can also see a small nodule behind the sclera which might be taken for an extension of the growth *viâ* the posterior ciliary vessels. Microscopical examination of the anterior part of the eye showed blocking of the canal of Schlemm by adhesion of the iris to the posterior surface of the cornea at the periphery of the A.C.: pigment in the canal of Schlemm; atrophy of the iris, ectropion of uveal pigment, and in some

PLATE IX.

Illustrates Mr. F. A. Williamson-Noble's paper on Intra-ocular Cholesterin Tumours (p. 189).

Fig. 1.—See text, p. 190.

Fig. 2.—See text, p. 191.

Fig. 1.

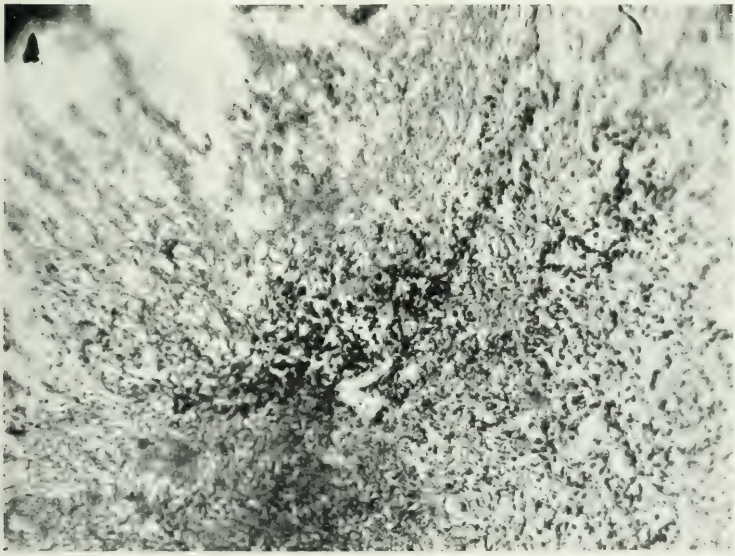

Fig. 2.

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of the sections a hæmorrhage, separating the pigment layer. There was also atrophy of the ciliary muscle and its processes.

The retina in the anterior part of the eye could just be recognised as the pars ciliaris: behind this, however, one can see the degenerative process beginning: a slide was shown taken at the junction of these two parts. Posteriorly the retina was scarcely recognisable: a slide showed what remained of it. It also showed some of the organised blood-clot lying immediately behind, with a little scattered pigment of hæmatogenous origin in its anterior layers. Further back there was a fairly abrupt transition from the organised blood-clot to a more cellular type of tissue, showing a certain amount of pigmentation and a superficial resemblance to a sarcoma of the choroid. The cells, however, were not so closely packed as in a sarcoma and were of a somewhat different type.

Pl. IX, fig. 2: This gives a fairly good representation of the appearances. Near the centre of the mass one notes the presence of a considerable number of pigmented cells. The others are polymorphic, some being round-cells and some of a very elongated spindle shape, suggesting that they are fibroblasts.

An interesting feature of this case is that the choroid is involved as well as the retina.

Another section was exhibited which showed on one side of the sclera the intra-ocular part of the mass of tissue, and on the other the extra-ocular part, to which I have already alluded. It consists of a more adult type of fibrous tissue than that which has formed inside the eye, arranged in bundles enclosing spaces which in the fresh state were probably filled by cholesterin. The only explanation I can offer for the formation of this mass is that it represents an old hæmorrhage which has undergone organisation with the formation of cholesterin crystals, and that the irritation of these has caused the formation of fibrous tissue.

The general appearances are strikingly similar to those exhibited in Mr. Mayou's second case, the slides of which I shall show presently. I have been able to find one somewhat similar case recorded in the *Transactions of the Ophthalmo-*

logical Society for 1916 by Mr. Beatson Hird, where there was an appearance of tumour formation owing to massive exudation into the retina consisting of fibrous and cellular portions. The choroid was not involved in any way, however, and appeared to be normal, whereas in this case the choroid seems to be definitely involved. As regards the pathology of this case, it would appear to be a somewhat aberrant form of retinitis, with massive exudation, as described by the late Mr. George Coats, though it differs from the usual type of this disease in two particulars, viz. :

(1) The involvement of the choroid.

(2) The presence of the extra-ocular mass.

These two points would favour the diagnosis of a choroidal sarcoma, though the cytology and general appearances are against this, and I should be glad to hear the opinion of members on this point.

Involvement of the choroid, of course, suggests tubercle, but there is no evidence of any tuberculous giant-cells.

Case 3.—The history is as follows :

A. B—, female, æt. 32 years. When 16 a glass bottle burst and cut her left eye. Under treatment for a month, and had an operation. Since the accident she has never seen with the left eye. Two months before Mr. Mayou's notes were made she knocked her eye against the corner of a cupboard. A week later she noticed a white patch in the lower part of the pupil, and did not think it had increased in size since then. V. : P.L. No ciliary injection ; t.n. Iris muddy ; iridectomy upwards. Pupil filled with capsule and old lens *débris*. Occupying the lower part of the A.C. was a whitish mass having some yellowish patches in it. In contact with cornea and iris and over it a fan-shaped series of straight vessels, breaking up into a number of fine branches. These were probably in the cornea. The tumour reached the angle of the A.C. except over a kidney-shaped area below.

The patient was a week under observation when the tumour appeared to increase in size and the eye was enucleated.

Pl. X. fig. 4 : The eye was divided antero-posteriorly. The retina was detached, showed multiple cystic degeneration,

PLATE X.

Illustrates Mr. F. A. Williamson-Noble's paper on Intra-ocular Cholesterin Tumours (*p.* 189).

Fig. 3. See text, p. 193.

Fig. 4.—See text, p. 192.

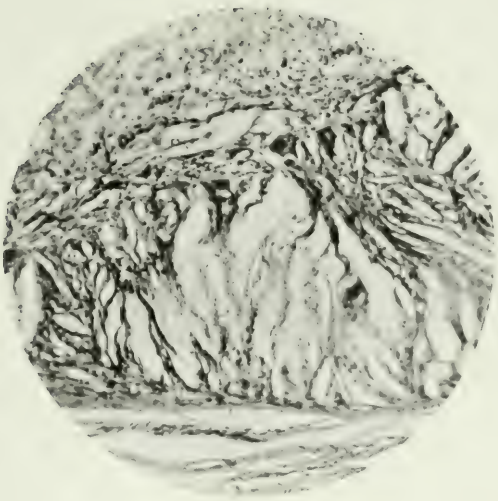

Fig. 3.

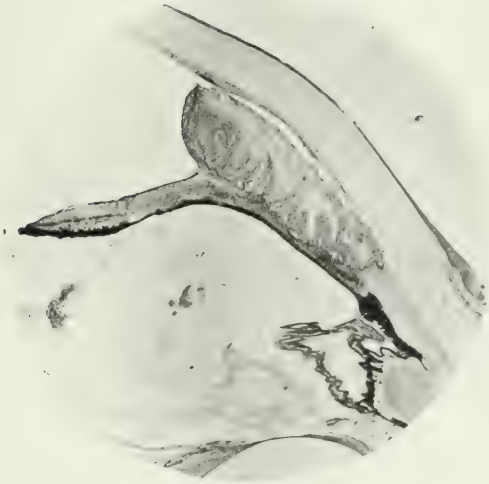

Fig. 4.

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and contained much fibrous tissue. The layers were not recognisable. The choroid was atrophic and contained fibrous and colloid patches and calcareous particles. A membrane consisting of capsule, lens *débris* and fibrous tissue lay behind the iris and pupil; the detached retina was adherent to it. The ciliary body showed signs of old cyclitis, and round-celled infiltration caused partly by the neighbourhood of the tumour and the pull of the detached retina. The iris in the neighbourhood of the tumour was pulled forward. There was marked thickening of the vessel walls and some round-celled infiltration and hæmorrhage. In the lower angle of the A.C. was the tumour, anteriorly adherent to and growing from the endothelium on the anterior surface of the iris. It was limited entirely by the lining membrane of the anterior chamber, and did not involve cornea, iris or ciliary body; the free margin had a pseudo-capsule of cells of the tumour. The parts of iris and cornea near the tumour, however, showed some round-celled exudations, and in the latter the vessels already alluded to were seen immediately in front of Descemet's membrane (Pl. X, fig. 3). The tumour itself consisted of elongated cells, loosely held together, which in the degenerate areas showed yellowish bodies in the cytoplasm. They grew from the endothelium of Descemet's membrane and the iris. Further out there was a formation of colloid bodies in this membrane.

This tumour then consisted of nothing but endothelial cells originating from the lining of the anterior chamber, showing no tendency to infiltration. No doubt it originated near the angle of the chamber and grew forward into it, since the two endothelial surfaces are connected by tumour substance.

Three diagnoses were possible—an endothelioma growing from the cells lining the anterior chamber, an organising blood-clot, or a mass of cholesterin crystals, giving rise to a new formation on the endothelium. Time did not permit of going into the pros and cons of these, but on weighing them up, they seemed to favour the last diagnosis.

Mr. M. S. Mayou said it was incorrect to attribute to him,

the speaker, entirely the cases mentioned; they came under his care as pathologist. One was Sir William Lister's case, and one was Mr. Cunningham's.

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11. *Superior paracentral scotoma after gunshot wound.*

By Gordon Holmes.

Dr. Gordon Holmes said that the circumstances under which the case was shown were that Mr. Juler told him, a few days ago, that he had a very interesting man with an inferior paracentral scotoma, and the speaker identified him as a case he had examined in France within a few days of the infliction of the wound. His present patient had a double superior paracentral scotoma, and he demonstrated the visual fields. There was also some defect on the left side, below the horizontal radius. Members had seen the course of the missile which caused the defect. It entered above the tip of the inion on the right side, and came out through the temporal convolution of the left side, and it must have therefore damaged the lower portions of the calcarine area on both sides, *i.e.* those portions below the lips of the calcarine fissure. The man had since been subject to epileptiform attacks, which occurred relatively frequently. The first warning of their onset was that the eyes became fixed, and the patient experienced flashes of light like a ball of fire; these appeared a little to the left of the point to which his eyes were directed; then they danced and receded, the vision became hazy, and he lost consciousness. The speaker saw the man in one of these attacks. His eyes were deviated to the left, and remained in that position during the attack.

These visual epileptiform seizures were very common after gunshot wounds. In most cases generalised seizures developed after a local aura; very few had visual aurae without sensory or motor phenomena.

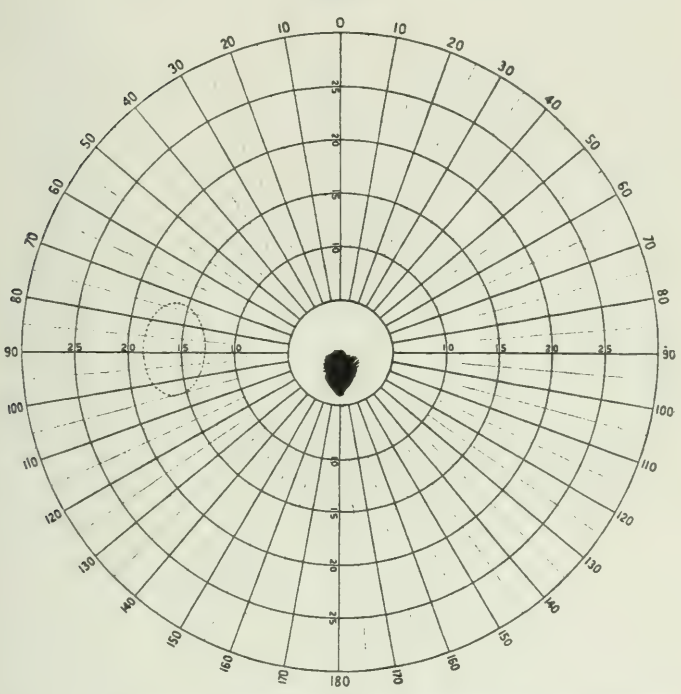
12. *Inferior paracentral scotoma after occipital wound.*

By F. A. Juler.

Henry B—, æt. 37 years. Wounded July, 1916.

Scar in occipital region, almost transverse, with thickening

Fig. 6.



Henry B—. February 15th, 1922. 1 mm. white at 330 mm. distance. Black = scotoma. Shaded = relative scotoma.

of underlying bone. Centre of scar 3·5 cm. above inion. Extent of scar 5 cm.; bony thickening, 2 cm. ong.

R.V. = $\frac{6}{9}$ slowly; L.V. = $\frac{6}{60}$, not improved (always defective).

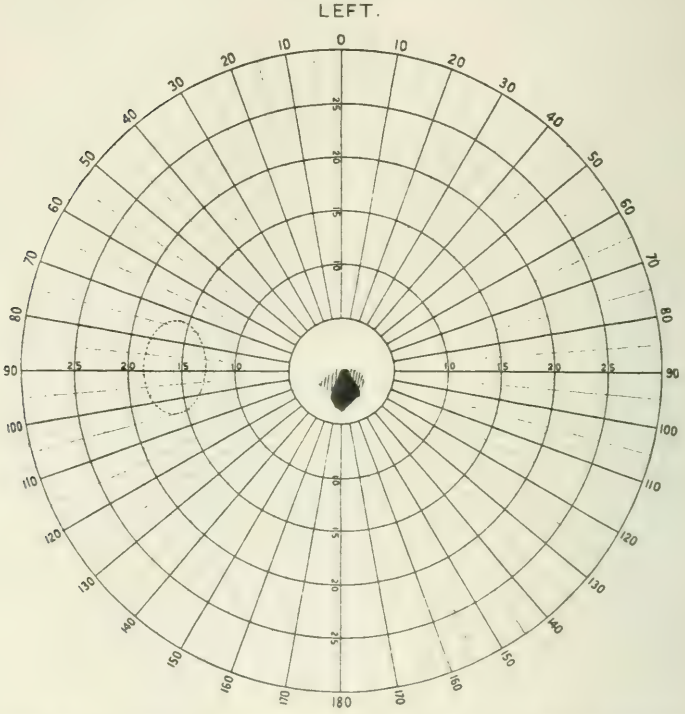
Refraction: R. sph. + 1 D.; L. sph. + 4, cyl. + 1.

Visual fields.—Both fields full in periphery to 10 mm. white.

Small inferior paracentral scotoma to white, extending from fixation point to 4°. Scotoma for red and green (2 mm.) exactly similar.

Fundi normal, save for slight "pseudo-neuritis."

Fig. 7.



Henry B—. February 15th, 1922. 1 mm. white at 330 mm. distance. Black = scotoma. Shaded = relative scotoma.

Case previously reported by Dr. Gordon Holmes (Case 2, *Brit. Journ. Ophth.*, July, 1918).

Dr. Gordon Holmes saw this man in France, too, and the scotoma mapped out by that gentleman then, closely corresponded with what the speaker had found independently since. There was no perforation track of the foreign body through the skull; there was just a contusion. The skiagram showed a slight depression of the bone over the calcarine area.

Dr. Gordon Holmes said the interest of this case was the persistence of the scotoma, almost unchanged, from the twelfth day after the receipt of the injury for six years. After periods from two to seven years he had been able to examine again a certain number of patients, whom he first saw in France within a few days after the receipt of their wounds, and in many of these very little change in the visual fields had occurred. In one case, that of a very intelligent man, there was a very small paracentral scotoma to the left and below the fixation point, coming to it but only extending 6° down. Sir William Lister and he examined the man early in 1915 five days after he received the wound, and when seen again this year exactly the same defect was present in the fields. That was very surprising, since motor and sensory phenomena due to acute injury of the cerebral cortex improve considerably in the course of time if the lesion is not extensive. These gunshot wounds, however, did not. He naturally excluded from these remarks those cases in which the defects were mainly due to acute septic processes, such as encephalitis or acute œdema of the brain. Therefore it must be concluded that the localisation of each separate part of the visual field within the visual cortex is very rigid, and the functions of any part which might be destroyed could not be replaced by that of any other part.

Mr. Traquair said he wished to make a plea concerning the examination of these cases as to whether any recovery took place. It was, that several test-objects should be used in the determination of the scotoma, so that one might ascertain whether there was any gradation of the edge of the scotoma. He suggested there might be a slight zone of amblyopia which might disappear after a time. The size of the test-objects and their distance from the screen should be recorded; only in that way could observations be made which were properly comparable at a later date, or in other hands. This feature of the examination had not been sufficiently recorded in the literature.

The President said another point which needed considera-

tion was, in penetrating injury, whether the foreign body traversed the brain substance. He saw a case similar to Mr. Juler's, in which there was nothing but a limited depression of the skull in the appropriate region. In one class of case the destruction was much greater than in the other. In one case the brain tissues might be temporarily deprived of all function, but there would probably ensue some restoration of function. In the other case the lesion might be so limited that there would be no damage beyond that of the tissues on which the bone was pressing.

Dr. Gordon Holmes said if a person received a sharp blow on the face, it did not matter very much provided the velocity of the striking body was sufficiently great whether it was withdrawn at once or not. This was true, too, in many of the cases of gunshot injury of the head. The phenomena are very rarely the result of mere compression of the brain, because the portions of bone driven in were impacted with such velocity that they destroyed at once the portions of brain substance they might later compress. The points raised by Mr. Traquair were very important. He, the speaker, always used graded test-objects as far as possible. He had, however, during the war made over 400 records of cases of gunshot wounds of the occipital lobe ; and, even if he had nothing else to do, it would not have been possible to test them all with graded test-objects. With many, however, he used different sized objects, both white and coloured. As in Mr. Juler's case, the margins of the scotoma were often extraordinarily sharp and frequently an object moving from the blind area appeared suddenly and plainly to the patient, just as if it had been brought into view from behind a screen. In most of these cases the contours of the scotomata for white and for colours were identical. In other cases—as in the one shown to-day—it was different, especially in the early stages, for in addition to actual destruction of brain there was often contusion of the surrounding tissue, and probably œdema and minute hæmorrhages. When the man he had exhibited regained consciousness he was unable to see the patient in the next bed, showing that his central

vision was completely lost, but within a short time he regained the amount of vision which he still had.

13. *A case of crossed hemiplegia (Foville's syndrome); tuberculoma pontis.*

By Leslie Paton.

E. H—, male, æt. 35 years. A patient in the National Hospital, Queen Square, under the care of Dr. Walsh.

Complaint.—Numbness and weakness all down right side. Loss of sense of direction in right hand, unable to touch things quickly unless he uses his sight at the same time.

Duration.—Eight months.

Past health.—Rheumatic fever (?) at 15. Treatment in a sanatorium eight years ago; otherwise felt quite healthy always. No history of venereal disease.

Family history.—Father died at 73—"stroke." Mother alive and well, over 70. Two brothers and four sisters, all stated to be healthy. One brother died in infancy. Patient married at 30; two children, both healthy.

Present illness.—Eight months ago woke up one morning to find he had a sensation of pins and needles all down the right side. On moving about, discovered he had a moderate degree of weakness in right arm and leg. Was out of work at the time, but was able to get work from November, 1921, to end of February, 1922. The numbness and weakness in right side had not changed. No sphincter trouble. Since end of February weakness in right side has increased and is more marked in arm than leg. Three weeks ago double vision commenced. Occasional dribbling of saliva from left corner of mouth. No headache. No pain anywhere. No difficulty in speech. Two or three times a week in last three months has experienced very slight "chattering" of lower jaw against upper, lasting an hour or two at a time.

Present condition.—Thin, poorly-nourished man of 35: has not been getting thinner. Speech normal. Memory and intelligence good. Marked internal strabismus left eye.

Alimentary system : teeth, fair condition ; tongue, furred. Appetite good ; no indigestion ; bowels regular ; abdomen *nil.*

Genito-urinary system : *Nil.*

Cardio-vascular system : Pulse regular in rate and rhythm ; tension low : Heart-sounds clear. No bruit heard. Cardiac dulness reaches nipple line on left.

Respiratory system : *Nil.*

Nervous system : Right-handed.

Cranial nerves : (1) *Nil* abnormal. (2) Acuity—R. $\frac{6}{9}$; L. $\frac{6}{12}$. Fields seem full by finger test. (3, 4, 6.) Pupils : R. larger than L. Both circular, central react to accommodation, direct light and consensually. R. eye : Position of rest, in slight outward deviation, but no further outward movement possible. Only very slight inward deviation possible (to mid plane), and done with great effort. Upward and downward movement normal. L. eye : In position of extreme inward deviation : upward and downward deviation normal. No other movement possible. Diplopia in all directions except when object looked at becomes obscured from L. eye by bridge of nose. No nystagmus. Slight ptosis left eyelid. (5) Corneal reflexes almost absent. Marked weakness temporal and masseter on left side. Diminution to cotton-wool very slight left side of face, more marked over cheek-bone than on forehead. Pin-prick better appreciated left than right—"did not feel like a prick on right side." Hot and cold better felt left, diminished right. (7) Slight weakness left side of mouth, seen on sustained action. Weakness in holding left eyelid closed. (8) Hearing good. Rinne + R. and L. (9–12) *Nil* abnormal.

Motor system : Arms –Grips L. stronger than R. Muscular strength fair, L. more than R. Tone equal and seems normal. Outstretched hands show light swing from normal on R. side only. Finger-thumb test and opening and closing fingers stiffly and slowly done R. side with accessory movements at elbow and shoulder. Bárány pointing moderate degree of outward deviation on R. side. Left side normal in all tests.

Trunk : No movement of umbilicus on raising head.

Legs: Right leg a little weaker than left. Extension at ankles more powerful L. than R. Tone feels equal in both legs. Co-ordination steadier L. than R., both accurate.

Sensory system—head and neck: Over scalp and anterior and lateral aspects of neck, pin-prick less well felt R. than L. Posterior aspect equal both sides. Cotton-wool equal front and lateral aspects posteriorly heavier L. than R. Cold and hot equal both sides of neck.

Arms: Cotton-wool a little better appreciated L. than R. Pin-prick sharper R. than L. Hot and cold equal both sides. Vibration sense absent R. side. Astereognosis R. hand. Localisation defective R. hand. Deep pressure absent R. fingers. Sense of position in space defective R. arm.

Trunk: Cotton-wool better appreciated L. than R. Pin-prick sharper R. than L., more "wide-spread" L. Cold and hot equal.

Legs: Cold more R. than L. Hot equal except on soles, where L. more than R. Cotton-wool, equal appreciation; both legs normal. Pinprick feels more diffuse L. leg, a little sharper on R. leg. Sense of position in space seems normal; also sense of passive movements at halluces. Localisation of toes touched better L. than R. Vibration less R. than L.

Reflexes: Arm-jerks all + both sides. Knee jerks, R. +, L. +. Ankle jerks R. +, L. +. Plantars ?. No ankle clonus. Abdominals, R. −, L. +.

Sphincters: *Nil.*

Spine: No scoliosis.

Joints: Arthropathy L. knee-joint.

R.O.D.: Disc edges even in their whole extent; no swelling. H.P. + 3; G.R. + 3.

L.O.D.: Some astigmatism ? H.P. + 3; G.R. + 3.

This case illustrates very beautifully the form of crossed hemiplegia usually known as "Foville's syndrome." The evidence of the symptoms indicates a lesion deep in the pontine region, involving the nucleus of the left sixth, the fibres of the left seventh, and at least parts of the left fifth nuclei, and also very definitely implicating the left mesial

fillet and some of the tracts lying external to the mesial fillet. There is just a possibility that in the region of the mesial fillet the mid-line has been crossed. The lesion is probably either a tuberculoma or a syringobulbia.

From the ocular point of view the most interesting feature of the case is the loss of lateral deviation of both eyes to the left, associated with the complete paralysis of the left external rectus.

Later history.—Since the meeting this patient has developed neuroparalytic keratitis in the left eye, and the lids have had to be sewn up to prevent further ulceration of the cornea. He has also developed a well-marked papillœdema with hæmorrhages. All the other symptoms have become more marked. There is little doubt that there is a tumour, probably tubercular, and rapidly growing and lying deeply in the pons.

The sensory loss is more marked all down the right side, with this exception—which I hope Dr. Gordon Holmes can explain—that he feels pin-pricks very much better on the side on which the general sensory weakness is most marked. It may be that the lesion has passed a little over and has caught the meso-fillet on the other side. Or there may be hyperæsthesia to pin-pricks on the right side. The general sensory sign is marked on the crossed side, except in the head and neck, where it is reversed. Therefore there is some motor weakness on the right side, possibly due to efferent disturbance. The flexor responses are doubtful.

Dr. Gordon Holmes asked whether the reflex movements of the eye to the left were affected. He only saw the case when passing through the ward. Then the patient was unable to move his eyes voluntarily to the left when told to do so; but if asked to fix his eyes directly in front and then rotate his head in the opposite direction his eyes went over well. If a finger which he fixed accurately was moved very slowly and uniformly to the left his eyes followed it easily, but if there was the slightest jerk so that the finger passed out of the macular vision the eyes immediately swung back to the right. Therefore some of the reflex deviations of the eyes

were preserved, though the voluntary deviation was lost. He suggested that the caloric vestibular tests should be applied in order to see whether the reflex deviations from the labyrinth were disturbed; he would be surprised if they were.

Such cases are not very rare. At Charing Cross Hospital he had recently a girl with the same condition bilaterally, When he first saw her she could not move her eyes to the right voluntarily, but the reflex and vestibular deviations to this side were normal. As she became worse she also lost voluntary movement to the other side, but both internal recti continued to act well in convergence. The case turned out to be one of acute disseminated sclerosis. More interesting phenomena were noticed as she recovered, since the dissociation between reflex and voluntary movements became even more prominent; she maintained her eyes well on the point fixed when the head was rotated, and when asked to keep her eyes on an object moving so slowly that it remained in macular vision, she followed it well even though she had no power of voluntary deviation to either side. In her acute stage he was able to demonstrate some of the Magnus de Keijn tests. He was lately asked to see a child who had suddenly become unconscious; her symptoms also were very interesting from the ocular point of view. While she was unconscious the eyes were a little deviated to one or other side, and she had extremely coarse persistent nystagmus to this side. Various movements of the head excited a number of movements of the eyes. On suddenly turning her head to the left, for instance, the eyes, which had been deviated to the left, swung over to the right to the full extent, and remained there for a considerable time. On tilting the head up the eyes deviated downwards, and slightly upwards when the head was flexed on the chest.

Mr. Leslie Paton, in reply, said he tried hard, a few days ago, to elicit the phenomenon referred to by turning the head, but it did not succeed. The patient had complete paralysis of the left external rectus, therefore the sixth nucleus must be involved. He thought it was now spreading up a

little more. It was only the motor fifth which was seriously involved; the sensory portion of the fifth was not so much implicated.

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14. *Capillary aneurysm on optic disc.*

By Leslie Paton.

A. E—, female, æt. 48 years. Case of hemiplegia, under the care of Dr. James Taylor, in the National Hospital, Queen Square. The patient was admitted in March, 1922, on account of pain, weakness and swelling in the left leg, weakness in the arms, and headache. The illness began in August, 1921. On examination the R.V.A. $=\frac{6}{18}$. L.V.A. $=\frac{6}{36}$. The pupils were active and fields full to finger tests. Ocular movements good. Slight nystagmus on lateral deviation. Right optic disc paler than normal. In the upper temporal quadrant was seen a small round red patch of the apparent diameter of 2 mm. It lay against a small artery running above it, and showed slight definite pulsation and a bright spot of reflex from its centre. The arteries in general were bright and thickened, and almost obliterated the veins where they crossed. The retina showed some tiny white spots close to the temporal margin of the disc.

Mr. Paton said it was the first time he had seen a capillary aneurysm in the disc itself. This case showed well all the characteristics which Gowers described in cases which Nettleship drew with great care. Gowers drew attention to the little glistening point, which was the distinguishing feature; it was still evident in this case. Some people had thought—he thought so himself—that they saw a little pulsation in that aneurysm, but he had not been able to convince himself of it absolutely. It much resembled some of the aneurysm cases which were described by Mr. Story as long ago as 1883, and which were figured in the third volume of the *Transactions*.

IX. APPENDIX.

The following were also brought before the Society :

(1) Tuberculosis of the Eye.—A pathological demonstration was held on the subject of "Tuberculosis of the Eye." The following gave demonstrations :

Sir John H. Parsons, C.B.E., F.R.S.
Prof. J. W. H. Eyre.
Mr. M. S. Mayou.
Mr. J. A. Ross.
Mr. Affleck Greeves.
Mr. Williamson-Noble (for Mr. H. Neame).

(2) Mr. N. Bishop Harman : A trial-frame gauge.

(3) Mr. N. B. R. Fleming : A cavernous angeioma of the lids.

(4) Mr. C. F. Harford : A case of glaucoma.

(5) Mr. N. B. R. Fleming : Bilateral tuberculosis of the lacrymal sac.

(6) Mr. B. Graves : A case of white spots on the lens after abscission of iris for traumatic prolapse.

REPORT OF COUNCIL.

The Council is glad to report the continued prosperity of the Society. There are 482 subscribing and 5 honorary members. During the Session 1921–22 twenty-one new members were elected. We announce with regret the death of five members: Sir G. Sims-Woodhead, who was an original member of the Society and a past Vice-President, Mr. C. Wray, Col. F. P. Maynard, Mr. G. F. C. Wallis, and Mr. W. J. Chambers. Five members have resigned.

In June, 1921, the Council nominated the following members to serve as representatives on the Council of British Ophthalmologists: Sir John Parsons, and Messrs. A. L. Whitehead, M. S. Mayou and A. B. Cridland.

An invitation, with the approval and promised support of the other ophthalmological societies of the Kingdom, has been conveyed by Mr. E. Treacher Collins to the International Congress at Washington to hold its next Congress in London in 1925.

Dr. G. E. de Schweinitz has been invited to deliver the Bowman Lecture before the Society in 1923, and the Council are glad to announce that he has accepted this invitation.

The Ninth Annual Congress was held at the Royal Society of Medicine, 1, Wimpole Street, on May 5th, 6th and 7th, 1921. The Bowman Lecture was delivered by Mr. Treacher Collins. There were discussions on the psychology of vision in health and disease, and the treatment of manifest concomitant strabismus. The Clinical Meeting was at St. Thomas's Hospital, and the thanks of the Society are due to the authorities of the Hospital for the facilities and hospitality granted. The meetings were well attended and the Congress was a success in every way.

The Transactions of the Ophthalmological Society of Copenhagen has been added to the list of exchanges.

During the last six months the post of Treasurer, vacant through the ill-health of Sir Arnold Lawson, has been filled by Sir William Lister, on the nomination of the Council, in accordance with Rule 14.

The Treasurer's Report and Statement of Accounts is appended.

HON. TREASURER'S REPORT.

From the account of the receipts and payments, it will be seen that the balance now in the bank is practically identical with that at the end of the preceding year, but this is not an indication that the financial state of the Society is all that could be desired.

Our chief liability is the cost of printing and illustrating the volume of the year's *Transactions*. Again it is very heavy. The cost of the Index made last year's *Transactions* particularly expensive, but the cost of producing this year's volume is more than £200 in excess of that for the volume of 1920.

It will be noted that the amount received for subscriptions amounts to £515 and the expenses of the *Transactions* to £696, which means that the latter cost the Society £181 more than its chief source of revenue. If the contributions of the Oxford Congress and the North of England Ophthalmological Society are added to the amount collected by subscriptions, the expense of the *Transactions* is still £144 more than it ought to be.

If it had not been for the exceptionally large and lucrative sale of past volumes our balance might well have been reduced to zero. It would seem extremely desirable therefore to see if steps could not be taken to reduce the cost without impairing the high standard which has been maintained in previous volumes.

The Illustrations Fund, as mentioned in the last report of the Hon. Treasurer, has stopped far short of the hopes and expectations of its promulgators. At the present total the amount of interest will be below £30 instead of the £70 that was hoped for. The fund is still open and a large number of members have not yet subscribed.

ARNOLD LAWSON,
Hon. Treas.

ACCOUNT OF THE RECEIPTS AND PAYMENTS OF THE OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM,

FOR THE YEAR ENDING MARCH 24TH, 1922.

RECEIPTS.

1921-22.	£	s.	d.	£	s.	d.
To Balance from last account				44	9	6
„ Subscriptions:						
Per Bank	282	10	0			
„ Collector	232	13	8			
				515	3	8
„ Dividends:						
East Indian Stock	14	2	2			
Ramsgate Corporation	15	6	0			
London County Council	9	0	0			
				38	8	2
„ Oxford Ophthalmological Congress	17	16	6			
„ North of England Ophthalmological Society	19	13	6			
				37	10	0
„ Sale of 'Transactions'				120	12	3
„ Illustrations				55	13	3
				£811	16	10

PAYMENTS.

1921-22.	£	s.	d.	£	s	d.
By Printing 'Transactions'	512	0	3			
„ Illustrations	148	3	0			
„ Sundries	36	15	10			
				696	19	1
„ Reporting				15	2	8
„ Royal Society of Medicine				30	7	6
„ Collector				25	1	2
„ Bank Charge				0	6	0
„ Balance at Bank				44	0	5
				£811	16	10

Examined and compared with Pass Book, Vouchers and Receipts, and found correct.

April 18th, 1922.

(*Signed*) M. L. HINE, M. H. WHITING } *Hon. Auditors.*

ILLUSTRATIONS ACCOUNTS.

	£	s.	d.	£	s.	d.
To Balance from last Account	52	5	2			
„ Transferred from Deposit	435	0	0			
„ Subscriptions	63	19	0			
				551	4	2
„ Interest on Deposit	6	3	11			
„ „ „ Funding Stock	8	16	2			
				15	0	1
				£566	4	3

	£	s.	d.
By Purchase of Stock	450	0	0
„ Balance in Hand	116	4	3
	£566	4	3

Examined and compared with Pass Book,
Vouchers and Receipts, and found correct.

April 18th, 1922.

(*Signed*) M. L. Hine, M. H. Whiting } *Hon. Auditors.*

OXFORD OPHTHALMOLOGICAL CONGRESS 1922.

ANNUAL MEETING HELD AT OXFORD ON JULY 6TH, 7TH & 8TH.

Master: SYDNEY STEPHENSON.

Past Master: The late R. W. DOYNE.

Deputy Master: P. H. ADAMS.

Hon. Treasurer: Sir ANDERSON CRITCHETT, Bart., K.C.V.O.

Hon. Secretary: BERNARD CRIDLAND.

REPORTS.

1. *Discussion on the significance of retinal hæmorrhages.*

By P. H. ADAMS.

IT may be roughly stated that hæmorrhages occur in the retina as the result of two chief causes: firstly, an altered condition of the blood itself, or secondly, a diseased condition of the walls of the blood-vessels.

When we come across a retinal hæmorrhage in the course of our routine fundus examination, I think we, as ophthalmic surgeons, at once realise that we have come across something of importance—something that cannot be casually dismissed as a trifle. We realise that here is an indication that the case is one which will require complete and careful examination, in order to arrive at a correct diagnosis of the cause of the condition. But it is not only the diagnosis of the cause that one is concerned with;

the thought of what does it mean at once presents itself, not perhaps so much as regards the sight of the patient, though this is of course important, but rather as regards the expectation of life.

Thus, the finding of a retinal hæmorrhage at once removes the case under examination from what might be called the optical side of our work, and transfers it to the medical side, and we are faced with the two problems of diagnosis and prognosis.

Diagnosis may be divided into local and general.

(1) *Local.*—A certain amount of information as to the cause of a hæmorrhage may be gained from the size, shape and situation of it.

Starting from before backwards.

Firstly, there is the preretinal or subhyaloid hæmorrhage. The blood is here poured out in front of the retina and assumes the familiar horizontal D shape; it apparently comes from an actual rupture of a small vein. As far as I know it has no special significance, and the prognosis, as regards vision, is good, as the retinal elements are undamaged and practically no trace is left when the blood has absorbed.

Next in order are the superficial striate or flame-shaped hæmorrhages which occur in the nerve-fibre layer of the retina, and are usually associated with arterio-sclerotic or renal conditions.

Then we come to the circular, mossy type of hæmorrhage. The best simile I can think of is, they resemble the mark left on a wall when a wet sponge has been thrown against it. These occur in the deeper layers of the retina and are most suggestive of diabetes. In this condition also one finds another type of hæmorrhage which is very typical, namely, the punctate hæmorrhage, a small dark round spot of blood, scattered generally over the fundus, about the size of a large pin's head.

The occurrence of these two types together, the superficial and the deep, may indicate the presence of both sugar and albumen in the urine of the patient.

Finally there is the pale-coloured hæmorrhage, or small white spot with red margin, suggestive of pernicious anæmia.

(2) *General.*—The occurrence of a retinal hæmorrhage discovered in a patient the victim of some general disease may be the means of leading the physician in charge to a correct diagnosis.

For instance, in cases of extreme anæmia it may be the determining factor which excludes the case from one of pure chlorosis and points to some more serious condition. For retinal hæmorrhages are found in more than 50 per cent. of progressive pernicious anæmias, and are also frequently observed in secondary anæmias, leukæmia, pseudo-leukæmia, purpura and scurvy, whereas they are very rare in pure chlorosis.

There is another small point that occurs to me in which the finding of a retinal hæmorrhage has put one on to the right diagnosis. I refer to cases of iritis of unknown cause, especially in elderly people. On examination of the retina one finds a few hæmorrhages, and on testing the urine sugar is discovered.

Again, the finding of retinal hæmorrhages on the surface of a detached retina is suggestive of the presence of a neoplasm behind and may be a valuable aid to diagnosis, especially when from the situation of the growth trans-illumination is of no value.

Prognosis.—This, again, may be divided into local, as it affects the eye and its vision, and general, as it affects life.

Local.—As regards the effect on the eye and its vision, the situation, whether macula or not, and the size and extent of the lesion, will cause obvious variations. If the macular region is free there is possibly no effect on the sight that the patient is conscious of, even though the condition may be gross, whereas a subhyaloid hæmorrhage in the macular region will cause an absolute central scotoma for the time being, which clears up completely in course of time.

General.—The prognosis as regards life is of considerably more importance. Of course one realises that it is not the hæmorrhages themselves that are of serious importance, but rather the changes in the walls of the vessels that allowed them to occur, or alternatively, the extreme alteration of the blood itself, yet because they are a definite result of these changes, it is impossible to separate the one from the other in a discussion on the subject.

A point of great importance, I believe, is, do retinal hæmorrhages occur in high blood-pressure, before the onset of disease of the blood-vessel walls? that is to say, in hyperpiesia, or the presclerotic stage of arterio-sclerosis? I cannot ever recall seeing a retinal hæmorrhage with high blood-pressure without some sign of actual degeneration of the blood-vessel wall. In connection with this, it is important to remember Foster Moore's observation that the blood-pressure in the retinal arteries is not raised in arterio-sclerosis, and that in his opinion hæmorrhages are not due to the high blood-pressure, but to the impairment of nutrition of the vessel walls and changes in the blood. If this is the case then, one must take it that the occurrence of a retinal hæmorrhage definitely shows that the case has passed beyond the more or less controllable one of hyperpiesia, and we are faced with a condition of true arterio-sclerosis.

However this may be, there is no doubt that some cases of arterio-sclerosis may show no signs at all in the retina; again there are certain signs generally recognised as indicating raised blood-pressure without actual disease of the vessel walls, such as heightened colour of the light reflex of the arteries, obliteration of the light reflex from the veins where the arteries cross them, and possibly slight pinching of the veins or alterations in their course, and finally very advanced conditions of retinal arterio-sclerosis occur in which there are no actual hæmorrhages at the time of examination.

There is another matter on which one would like to

have more definite information. Is there any explanation why one patient gets a venous thrombosis, another simple flame-shaped hæmorrhages, and a third arterio-sclerotic retinitis, all three showing definite signs of sclerosis of the arteries; and why should arterio-sclerotic retinitis occur in one eye as a rule, when the sclerotic changes occur in both eyes? Venous thrombosis, again, is a condition which may occur in comparatively young patients without obvious cause and without any signs of arterio-sclerosis. It was suggested that it was due to an alteration in the coagulability of the blood. Some years ago I examined a good many cases with this idea in view, but could not satisfy myself that there was anything permanently abnormal in the coagulation time of the blood, and I believe that this has been found to be a very inconstant figure, varying with the time of day, amount of exercise, food, and the length of time after it.

Of sixteen older cases of venous thrombosis that I have looked up, six cases occurred between 50 and 60 and ten between 60 and 70. Some died a few months after its occurrence, others lived for twenty years or more, so by itself one cannot say that venous thrombosis is of very definite prognostic value. It is the general condition of the patient, the condition of the heart, kidneys and blood-vessels, with which it is associated, that makes the difference.

Similarly, patients with arterio-sclerotic retinitis and striate hæmorrhages are known to live for many years, and in a paper which I had the honour of reading before this Congress several years ago I recorded many cases which lived on into the seventies and eighties in spite of retinal vascular lesions, the chief circumstances which affected the duration of life being whether the kidneys were involved or not. But one interesting point is, that in the majority of these cases when they do die, a vascular lesion is the cause of death.

The moral of this, then, is not to jump to a hasty conclusion that our patient is doomed to an early death

because we have found hæmorrhages in the retina, but to look on it simply as an indication for a full and complete examination of the general condition, and that only then can an accurate prognosis be made.

To pass on now to the question of diabetes. At present our knowledge of why some cases of diabetes have ocular complications, such as retinitis and hæmorrhages, and others do not is rather limited.

As ophthalmic surgeons, all cases that we see have these complications in some form or other, but as a matter of fact it is stated that it is only in 20 to 33 per cent. of cases that ocular conditions are found.

Hæmorrhages in the retina are, at any rate, not necessarily associated with the presence of sugar in the urine. All of us, I suppose, have come across cases of typical diabetic retinitis in a patient complaining of defective sight, and on inquiring into the medical history one has found that he or she says that they did have diabetes some years ago, that they were treated for it by their doctor and dieted for some years, but that now they were supposed to be cured of it, and that the last few specimens of urine that have been examined have been found to be sugar-free, and it is only since then that the sight has begun to fail.

It is, anyhow, a late symptom, but in spite of this many patients live for many years after its onset. Thus the prognosis is nothing like so serious as regards life as true nephritic retinitis, but the prognosis as regards recovery of vision is bad, the macular region is frequently involved, and the retinal changes persist in spite of treatment and possible general improvement.

In pernicious anæmia the occurrence of numerous small hæmorrhages followed by multiple ones and later by large extravasations of blood is said to indicate a serious prognosis.

Eales' disease or recurring vitreous hæmorrhage is usually found in young men, due to some disease of the blood-vessel walls, possibly with formation of small

circular aneurysms. It undoubtedly has a serious significance as regards the sight of the patient, but what is the cause? It has been suggested that it is due to tuberculous disease. I have recently had a case in hospital here, of a young man who went right through the war as a signaller, and was a volunteer fireman in civil life, whose sight suddenly failed in one eye from this condition the other day. I have had his blood tested by the complement fixation test to tubercle on two separate occasions, each time with a definite negative result and there was no other sign of tubercle about him.

Retinitis circinata is a somewhat rare condition and its cause at present not perfectly understood; it may occur associated with sclerotic changes in the retinal blood-vessels; at other times these are apparently normal. The actual circinate retinal condition is secondary to macular hæmorrhage; it has of itself no prognostic significance as regards life I believe, but central vision is usually permanently lost though the circinate patches may practically disappear.

Traumatic Hæmorrhage.

This may occur in the retina as the result of blows and concussion injuries. The cause is usually obvious.

The cases of real importance though, because easily overlooked, are the slight ones, such as I have seen caused by a flick from the lace of a football, or after a shot penetrating the orbit. These accidents may cause small retinal hæmorrhages right at the visible periphery of the retina, and sometimes associated with the escape of a drop of blood into the vitreous in the neighbourhood. If these cases are not carefully treated by rest in bed until the hæmorrhage is absorbed, they are a very fruitful source of detachment of the retina, which becomes noticeable some weeks after the injury and may lead to loss of sight of the eye.

Finally there are certain rare cases in which one comes across perhaps a single small flame-shaped hæmor-

rhage in the retina, during the routine examination of apparently healthy people, not of advanced years. These, I believe, may be due to strain, possibly from a cough, or constipation; they absorb quickly and as far as I know need not recur, and I belief mean no more than a subconjunctival hæmorrhage.

I should like to add that in trying to specify what a hæmorrhage means by its size, shape or situation, one is not trying to definitely make a diagnosis without taking other factors into consideration. I remember that at the Ophthalmological Society's discussion on diabetes a speaker remarked that anyone was a fool who diagnosed diabetes from a mere examination with the ophthalmoscope. I agree; but as an ophthalmologist I say we take a pride in trying to make our diagnosis first from the opthalmoscopic appearances; and one feels great satisfaction if, when one has examined and found hæmorrhages and retinitis, and has formed an opinion as to what the causative condition is, one then proceeds further to the full examination and finds that opinion confirmed. There is no doubt that the more one practises, the better are one's attempts to arrive at the diagnosis. Still, I admit, one never attempts to make a positive diagnosis by means of an ophthalmoscopic examination pure and simple.

ERNEST CLARKE:

I congratulate Mr. Adams on the excellent manner in which he has opened the discussion and the clear way he has expressed the salient facts, and I also congratulate the Council of this Ophthalmic Congress on selecting this subject, which is a most important one, and of which so little is written and known. There can be no doubt that hyperpiesis or hypertension is a very subtle state, and that it is always a sign of the *beginning* of a pathologic change. If permanent it is a certain sign of the presence of toxæmia, and even if no lesion of the heart or kidneys is discovered, it indicates some error in metabolism or

deficiency of elimination, either intestinal or urinary, or both, or the remains of a past poison, such as syphilis.

It has been said that in these cases the pulse-pressure, *i. e.* the difference between the systolic and diastolic pressure, rises much above normal, *i. e.* to 60 or 70 instead of 30, but I understand that this is now denied and it is not constant, and that what the physician really dreads is a great variation in the *systolic* pressure from day to day. But let us start at the beginning of things. Does the poison circulating in the blood first cause a thickening of the walls of the smaller arteries, resulting in an increased effort to pump the blood into the tissues, which, of course, eventually leads to higher blood-pressure? If so, then a later stage is degenerative changes in the intima caused by an insufficient blood supply to the parts, and possibly the local effect on the vessel walls of the poison circulating in the blood, and this in turn leads to local aneurysm, thrombosis and hæmorrhages.

It is stated that a high sphygmometer reading is, in a very large number of cases, *not* followed by retinal hæmorrhage; that a high reading in the brachial does not mean high pressure in the retinal arteries, and that a hæmorrhage is *not* a bursting of these arteries from the local pressure.

When a retinal hæmorrhage does not follow high blood-pressure this probably means that there has been enough pressure to supply sufficient blood to the parts, and the circulating poison is not powerful enough, and so degenerative changes in the vessels have not, so far, taken place. If this is the correct interpretation then we are interfering with Nature and doing harm by lowering the blood-pressure *directly*, unless it is unduly high, for by doing so we starve the tissues and help the degenerative changes.

So much for that theory. Now let us take the other. If the hyperpiesis is the primary trouble brought about by the toxins stimulating the adrenals, then the thickening

of the arterioles is a protective measure, and in its turn causes a vicious circle by increasing the blood-pressure, which is a natural sequence. Whichever theory is correct one must come to the conclusion that the blood-pressure, being itself a protective measure, must not be lowered directly, but we must do so by seeking and removing the *cause*—that is, we must remove the toxins. Fortunately for the patient any lowering of the blood-pressure directly —*e. g.* by bleeding—is only temporary.

Where does the oculist come in? Unfortunately he comes in very often at the end when the arterio-sclerosis is well established, and all he can do then is to warn and hope thus to prolong life a little. Whatever the patient's symptoms may be, whether due to changes in the blood or the vessel walls, a retinal hæmorrhage is in every case a danger-signal, and in most cases it is, to use a railway phrase, the "*home*" danger signal, and the patient must be told this and sent to a physician. Where the oculist can be useful is to give this warning *before* any hæmorrhage has occurred, and before the arterio-sclerosis is well established.

Thousands of people never have a doctor, and the only medical man who has a chance of examining them may be the oculist who is consulted for glasses.

This is his opportunity to benefit the patient. Most of these patients are 50 or older and the oculist should be on the look-out for the very earliest sign of arterio-sclerosis or of toxæmia, which may eventually lead to it; he can then easily warn his patient, and by *preventive* treatment save him many useful years of life.

One of the surest signs of the presence of toxæmia is a marked lowering of the accommodation power, and when this is accompanied by hypertension it is a fairly certain warning that arterio-sclerosis may be expected. In a very large number of these cases, as Sir Arbuthnot Lane has pointed out, the toxin is due to either intestinal stasis or pyorrhœa.

Again, a careful examination with the ophthalmoscope

will often make one suspect commencing changes. There may be nothing definite, but the vessels may be slightly more tortuous than normal, and here and there, where an artery crosses a vein, you can "spot" slight thickening of the artery walls. In such cases take the blood-pressure, and if it is raised, or if you do not take it yourself, but suspect it to be high, send the patient to a physician for a complete examination. Remember that the normal blood-pressure of a man of 60 is about 140.

Lastly, the oculist has it in his power to prevent retinal hæmorrhage by removing eye strain. Eye strain means increased work in the eye and this means bringing more blood to the part. This increased blood in diseased vessels may easily lead to a hæmorrhage.

Correct any error, however small, and as this class is generally presbyopic prescribe *bifocals* for constant use. Another very important point is that by removing eye-strain we increase the resisting power of the individual, and thus assist to eliminate any poison that may be present in the blood.

It should not be forgotten that physicians dread, in these patients, *hypotension* more than they do hypertension. If the vessels are diseased, thrombosis followed by hæmorrhage is very likely to occur, and what makes matters worse is that the whole resisting power of the individual is lowered. Here again *eye strain* must be eliminated.

In the foregoing remarks I have been dealing with retinal hæmorrhages as a sequel to arterio-sclerosis, and their significance is so serious because it points to the probability that the arteries of the brain are in the same diseased state as those of the retina, and we know to our grief how often this is the case. So many patients consult us too late, and end their life's pilgrimage very soon with a cerebral hæmrorhage.

The significance of retinal hæmorrhage in connection with diseased blood states where the arterial walls are unaffected may also be serious.

Hæmorrhages occurring in diabetes, glycosuria, leukæmia, anæmia, etc., may clear up, and the patient may be restored to complete health if the disease is not too far advanced.

Two years ago I saw a lady, æt. 52 years (sent to me by Dr. Benham), who had optic neuritis in both eyes with both fundi covered by hæmorrhages. Blood-pressure was normal. Dr. Buzzard diagnosed "toxæmia," and Dr. McCrae corroborated this and found the blood poisoned with streptococcus. Dr. James Taylor also saw her. By vaccination she completely recovered. I saw her three months ago when both fundi were normal, and the vision $\frac{6}{5}$ in both eyes, and she was perfectly well; if this condition had been due to diseased blood-vessels the patient would certainly now be in her grave.

Traumatic hæmorrhage in the retina, although it may be serious to the eye, is supposed not to have much significance as regards life. I do not think this is correct; in many cases the traumatism would not have produced hæmorrhage had the vessels been perfectly healthy. A year ago I saw a lady, æt. 58 years, who had fallen on the back of her head and lost vision of the right eye shortly afterwards; the right fundus below was covered with hæmorrhages, her blood-pressure was 180, and a careful examination revealed commencing arterio-sclerosis everywhere.

One fact seems to me undeniable—that if an oculist in the course of his examination discovers a retinal hæmorrhage, it is his bounden duty to refer the patient to a physician for a thorough overhauling, and, if necessary, medical treatment, and on his side the physician should never forget that he is only acting for the oculist.

Mr. T. Harrison Butler

said that he thought that most of the members had been brought up with the idea that practically all the conditions under discussion pointed to an early demise. When a student he was told—and he believed it for many years—

that when albuminuric retinitis appeared the patient died within two years, and the same was to be expected with retinal hæmorrhages of the flame-shaped type. Experience soon taught us that this idea was far from the truth, and the work of Mr. Adams definitely proved that there were many exceptions. Although this was an erroneous and exaggerated teaching, he thought that at the present time there was a tendency to minimise the importance of hæmorrhage; the pendulum had swung too far in the opposite direction. He would mention two illustrative cases. Ten years ago a young girl came to consult him about headache; nothing else. He noticed a small hæmorrhage near the macula in one eye. Investigation revealed the presence of marked polyuria. The urine was found to contain a trace of albumen. An experienced physician took an optimistic view of the case, but a fortnight later the girl became comatose and died without recovering consciousness. A case of this kind makes a lasting impression upon the memory.

A month ago he saw a lady, æt. 70 years, who was carrying out her ordinary household duties, and did not imagine that there was much the matter with her. He found albuminuric retinitis with flame-shaped hæmorrhages. He impressed upon her daughter that the matter was of serious import, and she arranged for her mother to give up her arduous house-work and come and live quietly with her. The lady died in uræmic coma three weeks after the interview.

In the past it was probably true that too much anxiety had been felt about these cases, but there was the liability that in the future they might not be treated seriously enough.

What ought we to say to the patient? He believed that Mr. Ernest Clarke had expressed the correct view in this matter. In many cases the first medical man these persons saw was the ophthalmologist, and they looked for some advice from him. Often when a general practitioner sent a patient he expected and was grateful for some

suggestion regarding treatment. In the case of a middle-aged lady he pointed out that the retinal condition was the red flag which pointed out the danger ahead. He advised her to give up the exciting life she was leading and lead a quiet life. This advice was not taken, and shortly she had a paralytic stroke, which did not kill her, but reduced her to a purely vegetative life. For years she lay in bed quite unconscious of the world around her. He had seen other cases equally bad in which proper treatment was adopted, and these individuals have lived useful lives for many years after the hæmorrhage.

He did not think that in diabetes hæmorrhages had any significance.

He had always imagined that the term "Eales' disease" referred to the vitreous hæmorrhage of adolescents, mainly of the male sex. Axenfeld had suggested that these hæmorrhages were caused by tuberculous disease of the retinal vessels, and he had treated cases with tuberculin with some success.

Dr. K. K. K. Lundsgaard (Copenhagen)

said the Congress had already heard in a very complete manner of the importance of hæmorrhages, therefore but little remained to be expressed. It was now known for certain that albuminuric retinitis was a very grave symptom, also that some retinal hæmorrhages drew attention to the imminence of glaucoma. With those exceptions, however, the significance of retinal hæmorrhages were not sufficiently known. Perhaps the field which first needed investigation from this standpoint was diabetes. By examining subjects of diabetes by means of the newest methods it would perhaps be possible to clear up some doubtful points. Since the examination of blood for the presence of sugar had become more common, it had become evident that patients might be suffering from diabetes mellitus without having sugar in the urine; it was even possible to find the sugar content of the blood

raised without that substance being detected in the urine. He thought it was possible to have a raised percentage of sugar in the blood in association with retinal hæmorrhages. His conclusion was that, if one wanted to find out the significance of retinitis diabetica, it was necessary to examine the blood for sugar, because diabetes could exist without glycosuria.

Mr. Foster Moore

said he believed that most retinal hæmorrhages were brought about by diapedesis, and were not the result of a gross rupture of the vessel wall. He had seen very large retinal hæmorrhages clear up entirely and leave no trace of their former presence, and it had not been possible afterwards to find any sign of a lesion of the vessels which traversed the area which previously was involved in the hæmorrhage.

With regard to primary thrombosis of the retinal veins in young subjects, he believed they were the result of a local phlebitis, as was made clear histologically by Coats many years ago. In most people who were getting on in life the disease was associated with arterio-sclerosis, and was due to changes in the vessel wall.

He believed that most hæmorrhages were primarily due to a change in the blood, and that vessel-wall changes were secondary to the blood changes. It seemed evident at any rate that in the blood diseases, leukæmia, pernicious anæmia and the secondary anæmias, in which hæmorrhages in the retina were very common, they were due to the blood changes, for there was no reason to think that there were any changes in the vessels. He believed it might be said that no patient died of pernicious anæmia without at some time developing retinal hæmorrhages.

A type of case in which the presence of a retinal hæmorrhage might be of the greatest value was when one was asked to say whether there was a beginning papill-œdema in a patient in whom perhaps headache and vomiting were prominent symptoms and the disc edges were a good

deal blurred. The question was whether the appearances were within the limits of physiological variation. It was often difficult, or sometimes impossible, to give a dogmatic opinion without seeing the patient again at a later date. It was in such cases that the presence of a hæmorrhage on the disc, however small, was of the greatest assistance.

In association with severe anæmias hæmorrhages were very common, but in addition it was not very rare to find white woolly-looking patches which were indistinguishable ophthalmoscopically from the cotton-wool patches of renal retinitis. He had not seen them mentioned, but when they were present with hæmorrhages renal retinitis was closely counterfeited. He did not refer to the star figure around the yellow spot.

He was glad to hear Mr. Adams tell the meeting that diabetic could be distinguished from renal retinitis. There were, of course, a good many cases where it was not possible to differentiate with the ophthalmoscope, but there were often features which were so characteristic that it was possible to predict with assurance that the patient was the subject of diabetes.

He thanked the author of the opening paper for his valuable contribution.

Dr. Gibson

thanked the Chairman for having given him the opportunity of listening to this discussion, because, speaking as a physician, he felt that ophthalmologists had a pull over physicians in that in the eye they were able to study living internal anatomy, and witness changes taking place in a way that physicians were not able to do.

He thought it was very seldom that hæmorrhage occurred from high blood-pressure, and the point he wanted to make was that this question of hæmorrhage was the same as that of arterio-sclerosis. Arterio-sclerosis had been looked upon as a disease of old age, but it was not necessarily that. As shown by *post-mortem* examination, the same process existed in the young as in people

over fifty years of age, and even those who were older might derive comfort from the fact that Prof. Turnbull is said to teach that arterio-sclerosis begins in early childhood. Certainly the same lesions were seen in the vessels in early childhood as were evident at a later stage of life; and he thought it might be useful to the Congress if he attempted a modern explanation of arterio-sclerosis.

Anatomically there were two distinct lesions. One was the degenerative lesion, such as was seen in the fatty degeneration of the intima of vessels. Secondly, there was an undoubted inflammatory lesion. One must identify the common process of arterio-sclerosis in some degree with an infection. He would relate it to the presence, in many normal people, of organisms in the blood. The passing by the kidney of organisms into the urine was a constant, a daily, phenomenon, but if it went on to a large amount the result was the production of lesions in the kidney. Bacteria gained entry into the blood by innumerable paths and were passed into the urine. Some of these may do damage to the vessel; and he looked upon arterio-sclerosis in no other sense than as a rash of the blood-vessel wall, the result, in most cases, of an irritation of the vessel wall by organisms. If that were true, the other lesions found, such as hæmorrhage, would fall into line and were understandable.

The question of thrombosis has been touched upon. That was a normal event in relation to the larger arterio-sclerotic patches in the aorta, and he only looked upon it as the effect of toxins on the blood, the destruction of cells, the setting free of thrombokinase, and the inevitable and consequent thrombus. Again, fibrosis of arteries was probably an irritation from toxins and bacteria which were circulating in the blood.

Those remarks led him to the question of treatment. The treatment consisted of an attempt to get rid of the toxins, which, he thought, were largely the result of infections. Pyorrhœa alveolaris was in great prominence at the moment, and he thought there was a clear connection

with arterio-sclerosis, and, in some cases, hæmorrhages and other arterio-sclerotic phenomena in the retina. Though he thought it was a factor, it was not always possible to associate a retinal hæmorrhage as caused by a faulty condition of the mouth. Other common infective conditions amenable to treatment were chronic appendicitis, chronic cholecystitis, or that mass of cases usually grouped under the term "intestinal toxæmia." For such treatment the general physician sent patients to a spa; there they lived in very good surroundings and the purgative waters produced a thorough intestinal lavage, which protected them against future depredations of this infecting process which originally caused the symptoms.

It had been a great pleasure to him to hear and take part in the discussion.

Mr. Ridley

said he wished to mention only two points. One was, that retinal hæmorrhages were very infrequent in hæmophilia. He did not ever remember seeing retinal hæmorrhages in a hæmophiliac unless they were due to accident.

The second point was, that he had come upon a number of cases in elderly people, about eighty years of age, who had minute punctate hæmorrhages, and when they occurred it seemed to be a bad sign in regard to continuance of life.

The Deputy Master,

answering Mr. Ridley, said he included hæmophilia in his abstract, but as he had never seen a case which showed hæmorrhages, and the reference was only copied out of a book, he did not mention it in the paper on purpose.

Mr. J. Jameson Evans

desired to ask about the occurrence of papilloedema in connection with chlorosis, because some years ago a very feasible explanation of this was given—that it was due to

thrombosis of the cavernous sinus. Associated with that he thought there was often a star figure at the macula, and he regarded that as a result of œdema of the retina. The question arose whether, in all diabetic cases with hæmorrhages, there was not arterio-sclerosis co-existing; certainly most of the advanced cases were associated with arterio-sclerosis.

Retinal hæmorrhages were either traumatic, toxic, or infective.

With regard to retinal hæmorrhages from infection, he supposed most of the retinal hæmorrhages were directly or indirectly infective in origin, though whether from toxins alone or from the infective emboli he would not like to say. He did not remember to have seen a case of retinal hæmorrhages from pyorrhœa, but when multiple abscesses existed, emboli of an infective character were sometimes seen in the retina and retinal hæmorrhages were set up. Pyorrhœa was a condition of free discharge of pus from the roots of teeth or from gums, and was not liable to produce emboli; it was probably from root abscesses that emboli proceeded, and retinal hæmorrhages were apt to occur.

Mr. Leighton Davies

desired to make brief reference to one point, namely, the presence of functional albuminuria in Eales' disease. He had seen only three or four cases of that disease, and in none of them was albuminuria found; he felt certain that functional albuminuria was not essentially a part of Eales' disease. The two varieties might be present in the same patient. One he remembered started with a large number of retinal hæmorrhages, and two or three years later had a sub-hyaloid hæmorrhage, which almost filled the vitreous. They all improved rapidly. This young man started the condition at 17, and for 10 years he had repeated hæmorrhages; yet to-day he was still carrying on his work as an accountant's clerk, therefore vision is not always seriously interfered with in these cases.

He wished to emphasise the point that high sphygmographic readings did not necessarily imply in the patient the presence of retinal hæmorrhages. Years ago he examined a large series of patients in a workhouse all of whom had cerebral apoplexy, and the blood-pressure reading ranged from 160 to 230 mm., yet in none of them did he find retinal hæmorrhages. That confirmed what had already been said.

Dr. Marion Gilchrist

said she could not resist the temptation to congratulate Mr. Adams on his valuable paper. She was much interested in the remark concerning the readiness with which in some people nothing more than a friendly grasp of the arm was followed by ecchymosis. A person with fibrositis got ecchymosis readily. A masseuse knew that a fibrositis patient would get ecchymosis at first under massage, but even hard massage later did not produce it.

She would like to take the opportunity to ask for a few hints as to treatment. As Mr. Harrison Butler had said, oculists were often asked to give advice for treatment by doctors who sent the patients. One was in regard to the most suitable diet. Her own inclination was towards a vegetarian diet pure and simple for these cases, but the difficulty was to choose a dietary which, while nourishing sufficiently, would not be too bulky. In regard to tea and coffee, too, there was a difficulty.

Dr. Stobie

expressed his gratification at being invited to attend the meeting. His association with Mr. Adams had been, to himself, a very valuable one. What had struck him most of all was, that very often the first medical man a person consulted was the ophthalmic surgeon, whatever the patient might be suffering from, *e.g.* diseases of the central nervous system, such as general paralysis of the insane, tabes, disseminated sclerosis, etc., diabetes mellitus, cardio-vascular or renal conditions. Therefore the ophthalmic

surgeon could no longer restrict himself to his own particular specialty; he had to be, in addition, a very expert physician. He had very much admired the spirit in which the ophthalmologist stepped in, where angels—in the shape of pathologists—feared to tread to explain these chemoses. That was all to the good. What the discussion seemed to show was, that they had to separate out the prognosis and treatment of these cases of retinal hæmorrhage into groups according to the ætiological cause at work. Mr. Harrison Butler had said—and he thought rightly—that there was now a tendency for the pendulum to swing too far in the opposite direction, and these hæmorrhages might be regarded too lightly. Everything seemed to depend on what particular disease the patient was suffering from. For instance, hæmorrhage might be associated with venous thrombosis in the eye following influenza, which was well known to produce venous thrombosis elsewhere, and the outlook in such a case was very different from that in infective endocarditis, where the lesion was probably a minute aneurysm; therefore the ophthalmic surgeon needed to be a fairly able physician, so as to be able to treat and prognose in these various conditions.

He would very much like to hear about tuberculous lesions in the eye causing hæmorrhages. There was a definite lesion in the arteries, yet one speaker said hæmorrhages were uncommon in tuberculosis. The difficulty was to find, when such existed, a tuberculous lesion in other parts of the body. An eye condition which was regarded as tuberculous was so seldom associated with a demonstrable tuberculous lesion elsewhere in the patient.

With regard to sphygmomanometric readings, it had been stated—and he had no reason to think otherwise—that hæmorrhages existed with low blood-pressure readings; but from the general point of view of the medical examination it was important to realise that there might be a very high systolic blood-pressure in people who had no arterial degeneration at all. For instance, numbers of soldiers

had been discharged from the army with what was known as "D.A.H.," men of twenty-five to thirty years of age. He had examined many of these men, and found blood-pressure readings of 170 to 180. One could chase up their systolic pressure. Therefore it was important in all cases to take the diastolic as well as the systolic reading, because in the cases where the systolic pressure was under nervous influences, the diastolic remained at its normal ratio. Occasionally also, a doubtful aortic regurgitant murmur could be confirmed by the very low diastolic reading in association with a high systolic figure.

Dr. D. Matheson Mackay

said he also had been much struck by the excellence of the opening paper, and wished to thank Mr. Adams for it.

With regard to prognosis when retinal hæmorrhages were found, one was frequently asked by a patient who came without having had any disease of which he or she was aware whether the hæmorrhages would go away. The attitude he took up was probably that taken up by most surgeons, namely, that this depended on the disease causing the hæmorrhage. If the patient was old, something often carried him off before the hæmorrhages disappeared. If the patient was young, the hæmorrhages usually disappeared unless there was underlying them some serious disease, which might, of course, be unrecognised or unexplained.

A question which arose out of some of the remarks made in the discussion was, Should the ophthalmic surgeon give advice to patients in regard to their general health? He considered that the ophthalmic surgeon should not. His own custom, when he saw a patient who might have some systemic disease, was to pass him on to a physician or general practitioner, though the latter unfortunately might, in some cases, take no interest in the patient. If a patient with general disease depended on the ophthalmic surgeon for treatment, he feared he would be relying on a weak reed. Even in the provinces he considered that what he had indicated should be the practice.

Mr. Thomson Henderson.

wished to support the remarks which had been made in connection with arterial diastolic pressure and its relation to hæmorrhages. By a method which he had discussed and brought before this Congress (*Trans. Ophth. Soc.*, 1914, xxxiv, p. 309), he was able to estimate by his pressure gauge the arterial diastolic blood-pressure in the retinal arteries of the eye, and in the few cases of hæmorrhages he had met with he had found such diastolic pressure raised. He had also found, in routine examination, patients with a raised retinal diastolic pressure in whom a raised sphygmometer reading was afterwards found by a physician.

Mr. Montagu Harston

said he had seen retinal hæmorrhages in cases of malaria, in which there had been undoubted damage to the vessel wall; and that was especially so where there had been a massive infection with malaria, particularly in what tropical people termed a "double infection." In the malignant cases the damage was due to the embolic influence of parasites during the process of sporulation. Hæmorrhages of this form were also encountered in dysentery.

He had been much interested in the discussion as to what one ought to do in these cases, namely, as to further treatment of the patient. He had often asked himself the question, "Why send the patient to a physician?" In his experience there was no good physician who was not an expert with the ophthalmoscope; neither had he met the ophthalmologist whom he would not consider a very excellent general physician.

Mr. John Hern

wished to utter a warning note against any tendency to consider retinal hæmorrhage as of no or very little moment. He spoke as one who had had forty years of experience. He had seen some of these cases thirty years

ago, and though he had been unable to find any cause for the hæmorrhage, he had found symptoms of disease coming on later. His position in the matter was that of Hughlings Jackson—that his conclusion as an old man was that cases he saw earlier and had regarded as of no moment came to him in later life with some serious disease of the nervous system.

Mr. Bernard Cridland

desired to know what was the significance of a subconjunctival hæmorrhage occurring in a comparatively young person. The text-books spoke generally of such as being of little or no moment. When they occurred in people of about forty years of age, should they be considered of serious import or not? He had in mind the case of a friend he had known seventeen years. His first professional knowledge of him was, that he came assuring him, the speaker, that he had a fly in his eye, which entered while he was cycling. It might have been so, but he had a small subconjunctival hæmorrhage. He came again a year or two afterwards, with the same story, and also a third time. Six years ago he came for a change of glasses, and a large retinal hæmorrhage was found, which, however, did not affect the macular region, and did not impair the vision. As his blood-pressure was 170 mm. he was sent to his own medical man and placed under treatment, and considerable improvement followed. When seen a year ago he had well-marked signs of arterio-sclerosis in the retina. Presumably in that case the subconjunctival hæmorrhages should have been regarded as early signs of the onset of arterio-sclerosis.

Mr. Traquair

said he would like to emphasise one point which was made by Dr. Lundsgaard—that too much attention should not be paid to the absence of sugar or of albumen in the urine in these cases. It seemed to be even more important to study what was retained in the circulation

than what was eliminated from it, and Dr. Lundsgaard, by examining the blood-sugar percentage, was able to say whether that was increased. There were methods of examining the blood in relation to the retention of other substances. He suggested that when ophthalmologists came across these cases in which they suspected there might be some such origin, and no sugar or albumen were found in the urine, they should not be satisfied until they had had the blood examined to make sure there was nothing being retained in the blood which ought not to be there.

Mr. Gray Clegg

said he had come across Eales' disease in a well-developed, healthy young woman, with repeated hæmorrhages. There was a rapid clearing up of the vision from only perception of fingers to normal on several occasions.

With regard to the occurrence of hæmorrhages in cases of detached retina he had such a case under observation, in which there were numerous, small hæmorrhages in one part of the detachment, and in another part there were white spots; and the question of a new growth entered his mind, and all the evidence seemed to confirm that view.

Mr. Philip Adams,

in reply, said he thought Mr. Thomson Henderson's pressure apparatus a very valuable one; he only wished it could be obtained more easily. He had been allowed to use Mr. Henderson's own instrument, and had been much impressed with it.

Mr. Evans had remarked upon the vascular disease and vessel changes seen in the fundus of the diabetic patient; these were sometimes very gross, and in his experience might be more marked in this disease than in any other. As an example of the danger of spot diagnosis, the last case of typical albuminuric retinitis he had seen was not due to that condition at all. He might have come to grief over it, because it was absolutely a typical text-book

case of albuminuric retinitis. He asked the sister-in-charge what was the state of the urine, and she said it had not been tested. He suggested that this should be done, and that was as far as he had committed himself. On the first testing there was a trace of albumen, but none subsequently. The man had a septic hip-joint, and was in a very bad state. After the leg had been amputated the retinitis cleared up completely, and the patient was still alive and well.

As to the proper course to pursue, he agreed with those who sent the patient to his medical practitioner, with a note explaining why. One was, however, at liberty to advise them generally for their good, because the treatment for the eye condition was the same as that for their general health. They should be told to avoid all preventable stooping, straining, and lifting of weights, as well as constipation. The rest should be left to the medical adviser.

2. *Discussion on methods of operating for cataract.*

Sir Anderson Critchett, Bart., K.C.V.O.

"The man who hesitates is lost." I have had recent cause to verify the truth of this statement, for when I was asked by our Council to open this discussion I should have taken the best course for you, and certainly for myself, if I had at once indicated that, using a Parliamentary formula, the reply was in the negative; but the invitation was conveyed to me in such gracious and seductive terms that I hesitated, and as a result I now find myself struggling under a burden of responsibility which I would willingly transfer to other shoulders.

It may perhaps be well that before dealing with present operative conditions we should take a brief glance at past history, and endeavour to trace the gradual process of evolution which has finally brought us to our modern methods. I therefore want you to accompany me on a

little retrospective mental journey, back to the time when as a small boy I saw Bowman do a flap extraction at Moorfields. I was too juvenile to be a competent critic, but I was much impressed by the perfect manipulation and the short time occupied, and I asked my father whether it was not a very easy thing to do. His reply was an amused smile and an emphatic shake of the head. The Moorfields staff must then have been near the parting of the ways, for at my next visit about a year later I was surprised to find that a revolution had taken place, that the king, as represented by the Beer's knife, had been dethroned, and that the operators were thrusting into the eye a strangely-shaped instrument which reminded me of a bricklayer's trowel. Ophthalmic surgeons had long been struggling with the difficulties and disabilities of the flap operation, and were ready to make trial of the new method suggested by Prof. Schuft. In England, however, operators could not accept the clumsy instrument which he had discovered for the removal of the lens, and more than one eminent ophthalmic surgeon placed the hall-mark of his inventive capacity upon the form of spoon which he deemed best suited to the operation. The merits of the new proceeding were much canvassed, and it was noticed that, while the previous risks of suppuration and extensive iritic prolapse were to a great extent avoided, fresh troubles arose to militate against the success of this form of operation. It was found that unless exceptional dexterity was displayed the passage of the spoon under the lens frequently caused rupture of the hyaloid membrane and loss of vitreous, so that in a comparatively short time the procedure had fallen into disrepute, and many operators, especially on the continent, were returning to their old love, the flap operation, when a paper read by my father in 1864 at the Congress at Heidelberg on the favourable results obtained by Bowman and himself with linear extraction attracted the attention of Von Graefe. He saw that the object to be gained was the power of making a curvilinear section of sufficient size to allow the ready

exit of the cataract without the introduction of a spoon or vectis into the eye, and he cut the Gordian knot of difficulty with the narrow needle knife which will always be associated with his name. As you all know, the use of this instrument has enabled an operator to make his incision in the situation and of the size which he deems best, and probably the ready adaptability of the operation to suit various opinions has proved a chief factor in its almost universal adoption. In the original combined operation as devised and practised by Von Graefe, the puncture, counterpuncture and final exit of the knife were all in the sclerotic; but it soon became manifest that this was allied to many possible disadvantages, so that at the International Ophthalmic Congress held in Paris in 1867, at which Von Graefe presided, my father was not surprised to hear that everybody was performing Graefe's operation with modification, and I think the same may be said of most of us at the present day. One of the most important modifications was that known as simple extraction, the Graefe knife being used to make the section, but the iris being left intact ; and I remember that my father one morning produced a mild sensation in the operating theatre at Moorfields by departing from the section which he was accustomed to make, and, inserting his knife opposite to the centre of the pupil, brought it out about midway between that point and the corneo-scleral margin. After lacerating the capsule he removed the cataract without performing an iridectomy. He had recently come from Brussels, where he had seen this operation performed by its inventor, Dr. Le Brun. During the next two years he sometimes used this method, and gradually made his line of emergence rather higher up, but experience proved that this method possessed several drawbacks, one of these being a tendency to the formation of extensive anterior synechiæ, the iris being not infrequently prolapsed and finally clamped between the edges of the wound. In addition to this serious defect, the effort to heal so extensive a corneal section proved more than some feeble

constitutions were able to compass, and even when union occurred without these complications there was often a considerable amount of traumatic astigmatism. The chief attraction which the operation offers is that it is very easy of performance.

Another purely corneal section belongs to the Leeds operation, and the latter was fully described by Mr. Pridgin Teale in one of the earlier Bowman Lectures. A knife of special shape is used, and the cut made by it results in a somewhat irregular opening in the cornea.

Another very ingenious but too complicated operation which deserves mention, though it could never be brought into general use, was one suggested by the late Dr. Bell Taylor, of Nottingham. Desiring to avoid prolapse and synechiæ, and at the same time to preserve the integrity of the pupil, he removed only a sufficient portion of the peripheral margin of the iris to permit the passage of the cataract. The result in successful cases was very perfect, the small iritic aperture being completely covered by the upper lid; but so much time was occupied in procuring the egress of the lens through this narrow and delicate channel, and so much technical skill was necessary to the successful performance of the operation, that it could never be commonly adopted.

It will, I think, be generally allowed that the ideal operation is the safe removal of the lens in its capsule. I first saw this performed by the elder of the brothers Pagenstecher at the Congress held in Paris in 1867. An unusually large incision, confined as nearly as possible to the corneo-scleral margin, was made downwards with a Graefe's knife, a liberal portion of iris was excised, a capacious spoon was then introduced beneath the lens, and the latter was removed in its capsule. It is interesting to note that two operations of the same type originated in India. In that suggested and practised by the late Mr. Macnamara more than forty years ago the operator introduced a spoon through the previously dilated pupil, and depressing the proximate edge of the lens, turned the

latter over into the spoon and removed it without rupture of the capsule. In more recent days we have been introduced to the strabismus hook method of Col. Smith, of Jullunder, and as we have read from time to time of his remarkable success we have felt that the tenth commandment was trembling in the balance. While I emphatically disclaim any intention to discount the merits of his exceptional skill, one cannot but envy him the possession of those calm submissive oriental patients. The latest additions to this type of operation come from South Africa and from Spain. When Lindsay Johnson, of Durban, was in England last year he described to me a novel instrument which he had invented for removing cataracts in their capsule. It consists of two (or possibly three) small shallow curved blunted hooks attached to one stem. After making an upward section he passes the instrument downwards across the anterior chamber till the hooks can be attached to the lower margin of the lens, and then with a gentle and steady upward drag he removes the cataract in its capsule. I was rather fascinated by his description of the operation, and I begged him to send me the little instrument on his return, but it has not yet arrived. I, however, recently received a letter from him in which he says—"I have been for two months to the Mauritius to operate on those of the inhabitants who wanted cataracts removed. I did them all by my method with absolute success. I never lose any vitreous, and the lens has to come out complete in its capsule; it cannot help itself. My operation is much simpler than Smith's, and I have described it in a paper which I have forwarded to Knapp for insertion in the *Archives of Ophthalmology*." We shall therefore soon have a detailed description of the method, and I confess that I am rather keen to try it.

At the Brighton meeting of the British Medical Association in 1886 the late Dr. Andrew, of Shrewsbury, described his operation for removal of the lens in its capsule with a spoon after division of the suspensory ligament.

I am glad that Col. Smith is to read a paper on the Spanish operation this afternoon, and Messrs. Weiss & Son have, I believe, brought the apparatus to this Congress, so you will be able to see its practical working. The latest reports from Moorfields are so favourable as to lead us to hope that Dr. Barraquer has really succeeded in cracking a fine nourishing Barcelona nut for us; but I fear that for some time to come the delicacy of the machinery and the difficulty of procuring and of maintaining the exact amount of electric current needed may in some degree militate against the general popularity of the method.

I, therefore, proceed to consider the operations which are in more common use amongst us at the present time, and we will begin with simple extraction. It is strange, but nevertheless true, that this plan of extraction has been adopted by some of the best and some of the worst operators. With reference to the latter, who shall, of course, be nameless, the late George Lawson used to aver that they chose it because they shirked the ordeal of the iridectomy. No such reason as this, however, could apply to such exceptionally dexterous manipulators as Snellen, Bell-Taylor, Gayet, de Wecker, Knapp, Trousseau, Stanford Morton and Devereux-Marshall. They were doubtless attracted by the optical and cosmetic results, and many of the sections which I have seen were so perfect that the king who failed to solve the problem of the apple and the dumpling would have been amply justified in expressing bewilderment as to how the cataract could have made its exit. An ideal operation, if we could assure ourselves that we should be able to remove completely all cortical substance, and that no prolapse of iris would occur. If this last-named catastrophe takes place it is almost impossible for the most skilful operator to make a clean and satisfactory iridectomy; and the difficulty respecting the cortex induced Knapp to invent a special cystotome, and caused that perfect operator David Little to discontinue the method after he had tried it in a hundred cases. As

I have already mentioned, my father succumbed to its fascination for nearly two years; but finally abandoned it when he realised all its drawbacks. The merits of simple extraction were advocated with much skill by Dr. Gayet, of Lyons, at the International Ophthalmic Congress held in Heidelberg in 1888, and in the after discussion his views were supported by Knapp and de Wecker. As I was then a junior member of the Congress I waited in the hope that some more doughty champion would rise to speak on behalf of the combined operation, but at length, as no one took up the challenge, I ventured to essay the task. After putting forward to the best of my ability the advantages of the plan on whose behalf I pleaded, I concluded with a personal appeal, which was I think a perfectly justifiable test, and it is one which I should like put to the proof on the present occasion. I reminded my distinguished colleagues that a time might possibly come to any of us when the operator must change his place and become the operatee, and I asked them to answer conscientiously whether under these circumstances they would select on their own behalf an operator who by a simple extraction might produce a finer cosmetic and possibly, but by no means certainly, a slightly better optical result, but who must steer them past the dangerous quicksands of prolapse and synechiæ; or whether they would not rather entrust themselves to the man who strove to secure the largest measure of useful vision for the greatest number, and who pinned his faith to extraction with iridectomy. I have reason to believe that my views were endorsed by a considerable majority of those present.

The size of the coloboma varies with different operators, the extremes being represented by John Couper, who made it from end to end of his section, and by Swanzy, who drifted so far in the opposite direction that he finally used a Tyrrel's hook for the iridectomy instead of iris forceps. I think we cannot do better than follow the direction given to us in our Latin Grammar days—"in medio tutissimus ibis."

Now a few words with reference to the merits or otherwise of preliminary iridectomy. This plan was initiated by my father in the following way. It sometimes happened that patients came to him who had undergone an unsuccessful operation for cataract on one eye, and all their hopes were centred on obtaining a more favourable result with the other eye. As a rule the cataract in the second eye was not mature, and my father, with a view to taking every possible precaution, decided to perform a preliminary iridectomy. He argued that by so doing he would eliminate the element of blood, and thus would have a clear anterior chamber for the removal of soft cortical substance. Also that he would avoid the very occasional but always tiresome complication when owing to a premature escape of aqueous or an involuntary squeeze on the part of the patient the iris falls over the edge of the knife. He found in addition that in very old and feeble patients the constitutional shock was lessened when the operation was divided into two stages. Besides these advantages the iridectomy tended to hasten legitimately the ripening of the cataract, and rendered the latter more speedily fit for extraction. As his experience of the operation increased he became convinced that his new plan gave a greater percentage of success than had resulted from the usual operation, and he maintained that under these circumstances it would be well to give the advantage of the proceeding in the case of the first eye dealt with, and not to reserve it only for the second eye. My own view is that one must be guided by circumstances. With a healthy patient and a ripe, firm cataract I am ready to make one complete operation, but under less favourable conditions I should advise preliminary iridectomy. I last year had cause to rejoice that I had done the latter in a case of a lady where she may be said to have had only half a remaining eye, for there were fairly extensive corneal nebulæ. The eye was a small one and deeply set, and I occupied at least a quarter of an hour in coaxing out the soft cortical substance, working the lower lid with my

finger, and waiting at intervals for the aqueous to re-secrete. The colleague who was kindly assisting me volunteered the remark that I could not possibly have cleared the anterior chamber successfully had I not had the invaluable advantage of the preliminary iridectomy.

It means two operations, and two visits to a hospital or nursing home, which certainly causes some patients inconvenience, but with cocaine and due antiseptic or aseptic precautions the added risk is very slight, while the advantages, as I have tried to show, may be very great. Perhaps the question of laceration of the capsule might be left for a separate discussion. Some operators, especially those who perform simple extraction, open the capsule on the passage of the knife across the anterior chamber in making their section. Some make a central crucial incision, and others follow Tweedy's suggestion and make it marginal. About the year 1878 my father tried opening the capsule with a fine needle shortly before the extraction, but it had this inconvenience—that it needed to be done at least an hour before the extraction to allow time for the anterior chamber to re-secrete aqueous for the section. The method has recently been re-introduced by Dr. Kirkpatrick, of Calcutta.

And now, before I conclude, may I venture to offer in all modesty a few words of friendly counsel to those of my colleagues who may have advanced only a few footsteps beyond the threshold of their career. In 1883 I gave a lecture at St. Mary's Hospital which was published soon afterwards in the *British Medical Journal*. Its objective was to advocate eclecticism in operations for cataract; and now, with nearly forty years' added experience, I want to preach to you from the same text to-day. I am inclined to doubt the judgment and the ultimate success of any young ophthalmic surgeon who says to me, "I always do so and so." Believe me, there should be no *always* in cataract operations. Ridding yourselves as far as possible of bias and prejudice you should approach each case from a free and independent standpoint, and be

ready to adopt the form of operation which experience has taught you will be most likely to meet the special conditions with which you have to deal. Let me give you an illustration of this which still lives in my memory, though many years have passed since the episode. I had gone by invitation to the Westminister Ophthalmic Hospital, where I saw that skilful ophthalmic surgeon and prince of good fellows Henry Power do some excellent muscular advancements. At the finish of these he expressed regret that he would only have one cataract extraction to show me, and after producing the patient he asked me what kind of operation he should perform. After a brief interval, during which I questioned and examined the patient, I said to my distinguished colleague, "As you kindly allow me to select the operation, I will be Irish enough to say that the one I choose is no operation at all—at any rate not yet." The patient, a septuagenarian, had been for some years in one of the old-time workhouses, and he was suffering from semi-starvation, insomnia and neurasthenia. Power at once fell in with my suggestion that the man should have a sojourn of some weeks at a convalescent home before the operation, and that when the latter was performed care should be taken to make a conjunctival flap at the finish of the section.

I recently read of a good instance of eclecticism on the part of our ever-alert colleague Maddox of Bournemouth. He had reason to believe that iritis was likely to ensue in a cataract extraction which he was making, so he decided to remove the lens in its capsule, and I am glad to say the result was successful.

In diabetic cases I would do a preliminary iridectomy, and at the second operation I would avoid a corneal section.

The principle of eclecticism also holds good with reference to patients who lose power of control. It will, I think, be generally admitted that it is better to have a friend who is good at a pinch than a cataract patient who is good at a squeeze, and unfortunately the latter species, though

less frequently met with than formerly, is not extinct. In dealing with this difficulty the operator has several alternatives; to avoid too peripheral a section, to adopt either the intercapsular method, or that with a conjunctival bridge, or, on occasions when a catastrophe seems inevitable, it may be wise to withdraw the knife, and, after a due interval, to make the extraction under a general anæsthetic.

I hope that I have not wearied you by my allusions to past, and in many instances antiquated, methods, but I was anxious to put in evidence the work of our predecessors, and to show what a wide choice of operations lies open to the modern ophthalmic surgeon. Of this I am well assured—that he will be the most successful operator who accomplishes his purpose in the gentlest possible manner, for in operations on the eye we have to deal with some of the most delicate structures in creation. Let us never forget that Dame Nature is a woman, and likes the last word, and always has it.

I have told you of my own operative predilections, and as they are founded on an experience which has had both latitude and longitude, I hope they may not be altogether valueless; but to-day I do not appear as a special pleader, I hold no brief for either plaintiff or defendant, for I have tried to the best of my ability to place the facts and the evidence impartially before the jury.

Lieut.-Col. Smith

said it had been intended that he was to read a paper by Dr. Barraquer, of Barcelona, but it had not arrived, therefore he would do the best he could under the circumstances.

He had been to see Dr. Barraquer. He, the speaker, was able to say he was fairly familiar with intracapsular extraction, and with the mechanics of that proceeding; he said "mechanical" because the whole of intracapsular extraction was a highly mechanical proceeding—one which could not be learned from reading a book, any more than shoeing a horse could be learned from reading

a veterinary book. He would like to see established in England a post-graduate school in ophthalmology, not for lectures, but as a mechanical teaching school, where men could learn the technical arts connected with the subject. He had himself done a good deal of post-graduate teaching, and had been able to select for operators the most promising of the students. They came to him specially to be taught cataract operations. The usual post-graduate teaching seemed to him designed to take away the innate confidence of the pupils. No one who had not established confidence in himself could hope to succeed as an operator. It was an Indian proverb that men generally over-estimated their own ability, but he believed it was Bacon's view that men usually under-estimated their own ability. The latter referred to the Briton, the former to the East Indian. He believed they were both very true of the respective peoples. East Indian educational people had told him, and his own observation confirmed it, that when the East Indian had acquired a certain amount of information he was apt to imagine he knew everything, and of course one would not get much further in teaching that class of person.

In the case of people who under-rated their own ability his observation was that, if the pupil had confidence established in himself, much more than half the work of teaching was accomplished. The pupil could then do much more and much better than he ever thought he could. The teaching of the present and of the past must be altered. The student is constantly warned of the thousand and one terrible accidents which may happen until he is so terrorised that he is shy, and always on the look-out for accidents which very seldom do happen. No system of lectures will ever make a surgeon. Nothing but the personal responsibility for the case is sufficient. Where the surgeon is concerned he has to acquire the manipulative dexterity necessary. This the junior surgeon cannot do on the living subject for the very good reason that the material is not availble; for ophthalmic operations there

is plenty of dead material, of which the aspirant should take full advantage.

The eye of the kitten six weeks old has a cornea of the same diameter as the human cornea. The kitten's eye could be fixed to something so as to raise the tension to approximately that of the human eye. The kitten could be killed by an injection of strychnine or cyanide of potassium, and the eye could be operated upon before the vitreous had time to liquefy. The lens in a young animal is very difficult to dislocate. The pig's eye was much stronger and denser than the human eye, but if one caught the pig's eye by the optic nerve end, and gave it a squeeze, then set it, an extraction intracapsularly could be beautifully done. Extractions and iridectomies should be practised on kittens' eyes until the action of the hand becomes almost automatic; every opportunity should be taken on the material available, and improvisation should be done until the necessary dexterity has been acquired, *i. e.* until the force of one's forearm had been eliminated; only the muscles used for writing should be employed in this operation. In the beginning the pupil requires an experienced operator to point out his mistakes until he thoroughly understands. Col. Smith exhibited the instrument used by Dr. Barraquer, including a suction device, brought into use by turning on the current. A criticism he had to make about it was, that in order to make the valve tight a certain muscular force was required, which tended to produce tremor in the hand. A suggestion made by him to an American operator was to have a longer tube, and have the valve managed by the foot; so as to relieve the hand of the strain of managing the valve while using the instrument.

Many of the troubles in connection with cataract extractions were related to control of the orbicularis muscle. If that muscle could be paralysed, one was operating, for all practical purposes, as on the dead body. Dr. Barraquer injected into the vicinity of the orbicularis a novocaine solution. The speaker had since met Major

Barnett, of the Indian Medical Service, who used as much as 40, 50, even 60 c.c. of a ·5 per cent. solution of novocaine from fifteen minutes to an hour beforehand. To get the full effect of the drug, Col. Smith thought it should be given an hour to act. Major Barnett injected it under the eye, the side of the nose and the region of the supra-orbital nerve, and over the whole region of the orbicularis—first a superficial set of injections, then a deeper set. That gentleman said it took effect more quickly if, when he had done the injection, he slipped in a strong speculum, which, presumably, somewhat obstructed the venous return. The amount used at Barcelona was very small, and the effect so light that one scarcely knew whether it was merely the difference between a good patient and a bad one. A great reputation awaited the man who found a means of keeping the orbicularis paralysed for seven days; then cataract extractions could be done without hesitation and without iridectomy. Prolapse of the iris was, he considered, in every case due to the patient, consciously or unconsciously, squeezing with his orbicularis in the days immediately succeeding the operation.

The patients he saw in Spain were heavily under a mydriatic, one which could be counteracted by eserine, and an ordinary retractor was used for holding the patient's eyelid against the brow. The incision Barraquer made was one of 180°, commencing in the sclero-cornea, and there was a kind of conjunctival flap. Less than 180° was not safe, more did not give the operator more room; if more room was wanted one should go deeper. Barraquer did what was called a button-hole iridectomy. In his, the speaker's observation a button-hole iridectomy was generally a complete iridectomy when the patient was leaving the hospital, or perhaps before he had left the operating table.

The instrument he showed had got so little suction power that though it would catch hold, it might or it might not retain that hold; it might lose its grip when

the lens was dislocated, and then one could not adjust it in position a second time without sucking the vitreous out of the eye. There seemed to be no reason why the suction employed should not be ten times that of this instrument. Indian patients were said to be very placid and good subjects for operation, but he had sometimes found it necessary to give them a general anæsthetic. He had found that the less one asked the patient to do, the better he would behave. At Barraquer's suggestion, the speaker did in Barcelona two operations by his own Indian method, and the lenses came out as easily as in Barraquer's cases, and with much simpler apparatus, and that authority agreed that in the hands of experts there was nothing to choose between the two methods. In Col. Smith's opinion, however, there was a good deal to choose between the methods. By Barraquer's method one had that complicated machine to keep in order, and there was so much about it that was liable to go wrong. He recently wrote a paper for the *British Journal of Ophthalmology*, having learnt that a considerable proportion of cases done with Dr. Barraquer's instrument had the complication of retinal detachment. In that paper he pointed out that it was probably due to the lens being simply hauled out. This applied a vacuum through the lens to the vitreous and retina. He pointed out that in any method of intracapsular extraction, *pressure* should be applied to cause the lens to rotate on its transverse axis until the suspensory ligament was partially separated from the lens before attempting to extract it. When the suspensory ligament had gone at one point, the completion of its separation was easy. In his own method that pressure was applied through the cornea on the lower border of the lens. When dislocation was partial the direction of the pressure was altered to keep it always at right angles to the plane of the lens, which was thus expelled from the wound by a continuation of the rotation on its transverse axis. He thought Barraquer's proceeding should be to insert his spoon horizontally from the side, get hold of the lower

border of the lens and dislocate in a similar manner by applying traction. When dislocated he could swing it out of the wound by rotating the spoon between the thumb and forefinger without fear of applying any vacuum to the vitreous and retina. Since the speaker wrote the paper he had referred to, he had heard that Barraquer took hold of the lens by its lower third and pulled it forward to make a somersault. That could not be done without making it rotate on its transverse axis. He would succeed equally well if he adopted the procedure just described.

That one could avoid occasionally having escape of vitreous and prolapse of iris he did not believe; all operators had accidents in operating for cataract.

He believed that the most interesting paper on the capsulotomy operation in modern times was that by Prof. Parker, of Detroit, at the 1921 Annual Meeting of the American Medical Association, at Boston. His total experience was about 1400 cataracts by the capsulotomy method, and his cases of escape of vitreous numbered about 14 per cent. When Col. Smith was asked to speak on the paper he complimented the author on his paper, and said the statement was clearly that of an honest man, as many operators would not have admitted having had escape of vitreous in so many cases. The 14 per cent. was probably due to his fear of leaving any lens matter behind, which was greater than his fear of the escape of a drop of vitreous, and he afterwards told the speaker that this was precisely his reason. So it will be seen that there can be escape of vitreous by the capsulotomy method.

(Col. Smith then demonstrated the use of the apparatus.) Barraquer's instrument, he said, cost £50, but there was no reason why a London maker should not manufacture them at a profit if he sold them at £10. He could not quite credit a statement that no escape of vitreous followed this method. He knew that prolapse of iris followed exceptionally frequently any operation without iridectomy

and he did not think the button-hole helped. He had seen extraction of cataract in capsule done by Czermac's method, which would require a larger incision than Dr. Lundsgaard's method. He had done a number of these himself. It meant snipping the sclero-cornea 180° with scissors. His experience had been that the conjunctiva cemented down promptly; and it was one of his objections to the conjunctival bridge. When one looked at the case next day, when the effect of the adrenalin had passed off, considerable blood was seen in the anterior chamber. The cementing down of the conjunctiva secured the full normal tension of the aqueous almost immediately, and that meant that the sclero-corneal wound was propped apart and a prolapse of iris got into that, and this was the worst form of prolapse of iris.

Dr. K. K. K. Lundsgaard.

The infections incident on operations for cataract are practically all of ectogenous origin. Endogenous infection may doubtless occur at rare intervals, but as it cannot be recognised as endogenous, we had better not reckon with it. That endogenous infection should primarily originate from the teeth has, as far as I can see, not been proved, and is little probable, and I for one never examine the teeth of my patients prior to operation.

As all my endeavours have been aimed at eliminating the chances of micro-organisms penetrating into the wound, and as I acknowledge that even the most careful antiseptic method provides no guarantee in this respect, because the conjunctival sac cannot be made sterile, I have attempted subconjunctival operation in order to work as far as possible in sterile tissue, and to be able, after the operation, to cover the wound *completely* with a conjunctival flap.

If a conjunctival flap is to cover the wound completely, it must of course be made before the sclero-corneal incision, otherwise the operation cannot be guaranteed to be subconjunctival. A true subconjunctival operation was

devised by Czermak in 1903. By undermining the conjunctiva he contrived a subconjunctival pocket, inside which he performed the many complicated manipulations necessary to remove the lens. Technically this operation was no doubt a triumph, but no further collection of cases has been published to illustrate its practical value, and not many ophthalmic surgeons have followed Czermak's method.

I omit Kuhnt and his successors' method, as not being free from dangers of infection.

Some years ago my friend Prof. Groenholm, in Helsingfors, in order to obtain a good even flap, made it with scissors, so that its base was the limbus. During the operation the flap was kept pulled down by means of fixation forceps, so that its attachment was visible during the performance of the section. Groenholm was at first well satisfied with the method; afterwards, however, it appeared that the big detached flap did not always keep safely in position, but might sometimes afterwards become folded down over the cornea, and in some cases infection occurred. After these experiences Groenholm soon gave up the method and tried a new one, in which he only loosened a flap corresponding to the temporal half of the wound; on the nasal side he leaves a bridge, which he has fashioned beforehand by undermining, but his Graefe's knife emerges outside this bridge, so that a small part of the wound is uncovered. Groenholm has performed this operation forty times.

In the course of one year and nine months I have practised the subconjunctival operation, using a technique almost corresponding to the one first used and afterwards abandoned by Groenholm. I have endeavoured to work exclusively in sterile tissue, and to obtain a really effective cover for the operation wound. That the technique has failed now and then I willingly admit, but as it appeared to me that Groenholm's excellent method was hampered with some minor faults (its nasal side towards the lacrymal sac is uncovered), I prefer the method described below.

In my operation I always use dry sterilised instruments. The hands of the operator and assistant are likewise thoroughly dried, care being also taken to remove all fluid from the eye subsequent to its irrigation, and dry sterile gauze is used exclusively during operation. I lay some stress upon these precautions, both as regards prevention of the spread of infection, and because I believe that the conjunctival flap will adhere far better to the wound when its inside has not been in contact with foreign fluids. I realise, however, that the spread of fluid cannot be entirely prevented because the accumulation of tears, which often takes place during a more protracted operation, may compromise the endeavour of keeping the operation field dry, just as the outflowing of the aqueous humour also counteracts this effort, but as far as possible the moisture is sucked up by means of dry cotton-wool plugs.

The lid retractor having been introduced, a small area of conjunctiva, situated 3–4 mm. above the limbus in the vertical meridian, is cauterised by means of a platinum rod, heated over a spirit-lamp. The small white area appearing after the cauterisation is now seized by a very fine anatomical forceps and an aperture is made by means of scissors.

The conjunctival flap is now formed by a blunt-pointed knife introduced through the hole, cutting from within outwards. The flap, dissected in this way, is seized at its outer side by fine anatomical forceps, and lifted up, whilst it is dissected with scissors towards the limbus. The flap is now pulled downwards over the cornea so that it covers its upper part; by this means a sterile operation field is obtained, consisting downwards of the inner side of the conjunctival flap, and upwards of the exposed sclerotic.

Now the eye is fixed at the limbus below.

The incision with the Graefe's knife is the most difficult part of the operation. It is essential to have a trained assistant, who can raise the conjunctival flap with the forceps in case it impedes the operator's view of the anterior chamber. Iridectomy is usually performed.

Cystotomy and expression of the lens involve no difficulties. The lens remnants may be pressed out in the usual manner, and should be carefully removed from the sterile area by means of sterile gauze. The iris can now be replaced, and the wound area having been cleansed of blood, the conjunctival flap is replaced and pressed gently against the sclerotic for about half a minute, after which time it usually will adhere very firmly, so that the eyelid can be closed over it without causing its displacement.

A binocular dressing is used for twenty-four hours. On changing the dressing the next day the anterior chamber has always been formed, and, indeed, this has also been the case when I removed the dressing a few hours after the operation to see that the flap was in position, which I did some few times shortly after the introduction of the method.

As a rule the patient had no pain, and the eye usually kept fairly free of injection, the flap adhering flatly to the underlying tissues; sometimes, however, it would bulge in the course of a few days with slight hæmorrhage and protrude rather prominently from the edges of the flap, which are already adherent.

I have performed the subconjunctival operation 56 times, of which 46 were simple and 10 complicated cases of cataract. In some cases I have not used the subconjunctival method, because I have feared that the patient's restlessness, inability to look downwards or tendency to squeeze made the more complicated operation inadvisable. In some cases, too, I avoided the method, because my usual assistant was absent, so that I had to work with a casual assistant, who was not familiar with the method. It is, however, my belief that it will be possible to apply the method for practically all cases when one gets accustomed to it, and more especially if paralysis of the orbicularis by novocaine injection at the outer corner of the eyelid is applied to those patients who show the dangerous tendency to squeeze. I did so in three of the mentioned cases.

In some cases the conjunctival flap was damaged from the point of the knife emerging in front of it; this happened four times. In this case a small linear area will remain uncovered at the nasal extremity.

The case is worse if the central flap is injured during the incision. In such cases the operation fails in so far as the middle of the wound is deprived of protection.

I have been fortunate enough in the cases here dealt with to escape those other troubles, which can only partly be evaded if a Graefe's knife be used, but which I dare say could be prevented, if it proves possible to combine the subconjunctival method and operation with a Jaeger's knife. This question I will leave here.

In three cases of complicated cataract—in one of which the lens was subluxated—loss of vitreous occurred, and the lens had to be removed by means of a Reissinger's forceps.

Displacement of the conjunctival flap happened twice. The first case occurred at a time when I placed the utmost confidence in its power to adhere, so I took no special precautions. The patient had very protruding eyes. On changing the dressing twenty-four hours after the operation the flap was displaced downwards over the cornea, so that the wound was entirely exposed. Nevertheless the anterior chamber was well formed. In the other case there was only a partial displacement of the flap. In order to avoid displacement I believe it to be essential not to make the flap too broad, for in that case it will tend to become everted. In one case of prolapse of the vitreous sutures were necessary. In one case with loss of vitreous the wound was infected, because the vitreous prevented the flap from remaining in position and the eye was lost.

Hæmorrhage into the anterior chamber two to four days after operation occurred in four cases, in each case after the patient had received permission to get up. In all these cases the flap was reddish black and bulging, and in two of the cases blood was seen hanging down into

the anterior chamber from the flap—the best proof that the scleral wound was open and that the essential closing first takes place at the margins of the conjunctival flap.* This complication did not cause other difficulties than a prolonged after-treatment. Good vision was ultimately obtained.

Infection of the wound or more deeply situated parts only happened in the case mentioned above, and never happened in Groenholm's cases. Iritis did not occur; in two cases, however, it was possible to see some isolated fine vessels in the iris, and in two other cases there were some few fine deposits on Descemet's membrane which soon disappeared.

Prolapse of the iris occurred in four cases: one in which I had purposely omitted performing iridectomy, and in two cases where iridectomy had been performed, a slight prolapse of the iris occurred secondarily.

Among the operated cases there were three whose conjunctivæ were infected with pneumococci, in which I failed to sterilise the conjunctiva beforehand. In one of these cases I had previously operated on the other eye, which was lost by panophthalmitis. The subconjunctival operation of the second eye ran an uneventful course, and good vision was established. A good indication as to the course of the operation and after-treatment is the day of discharge of the patient. I do not recollect ever before to have dismissed patients operated upon for cataract earlier than the fourteenth day after operation, while now I dismiss them with the same feeling of confidence on the eleventh or twelfth day, and I am inclined to believe that they might often be dismissed without any risk still earlier, if the conjunctival wound is healed, perhaps on the ninth or tenth day.

The material upon which this paper has been based is not large, but I dare say is large enough for me to venture to recommend you to try the subconjunctival

* The scleral wound under the flap will keep open (apart from the mechanical, chemical adhesion) until the formation of the cicatricial tissue sets in after four days.

method of operating, and more especially I venture to do so, because Groenholm's and my own material point in the same direction, and together we have about one hundred cases at our disposal. The technique is somewhat more difficult than the one usually applied, but this is compensated by the feeling of confidence one has after the operation, a feeling which is absent when you do not use the conjunctival flap.

The chances of infection are no doubt reduced to a minimum by the method, the risk of glaucoma being hereby almost excluded, as well as the danger arising from inclusion of iris and capsule in the wound when this is not covered by a conjunctival flap.

Mr. T. Harrison Butler.

As the time is getting late I shall treat the subject very shortly. I have described the details of the operation in a paper which will appear in the next volume of the *Transactions of the Ophthalmological Society*, therefore I shall now confine myself to the broad aspects of the subject, laying stress upon the disadvantages of a conjunctival pouch. These have been ably set forth by Lieut.-Col. Smith and I agree with all that he has told you about conjunctival flaps and pouches.

I do not think that anyone will controvert the opinion that we should so order our cataract extraction that the lens is easily delivered with the least possible trauma and pressure. Any procedure which calls for increased force in delivery is to be avoided. Success depends largely in allowing the lens to escape easily. If the incision be large enough and the manipulations correctly applied the lens should appear with practically no pressure. The word "expression" in this connection is an abomination; the lens should be coaxed out—almost charmed out—by the slightest pressure at the bottom of the cornea. As soon as about one-third of the circumference appears then I gently draw it out with a lens hook. Many of the new methods do not allow of this persuasion, and for this

reason I reject them. There is not the slightest doubt that the delivery of a lens under a conjunctival bridge calls for considerably more force than that necessary in the open operation, and for this reason it is self condemned. Under exceptional circumstances when we are faced with a straining patient then we are justified in choosing the bridge method.

To perform the operation with a bridge I employ a Smith knife. I prefer it because we are unlikely to introduce it backwards; because the two sides are quite different in appearance; because the incision corresponds with the back of the knife, which is a straight line; and finally because it goes in more easily than the more obtuse Graefe.

The incision commences about 1·5 mm. from the semi-diameter. There is nothing against this large incision and all for it. The knife travels in the limbal region and emerges under the conjunctiva. The incision is now carried up as far as possible and the bridge is formed. If necessary the two side incisions in the conjunctiva are prolonged upwards with squint scissors. It is wise to make the opening equal on both sides. It may be easier for the lens to escape on one side rather than the other. Again, if the two limbs are unequal there is a strong tendency to oblique astigmatism.

Within a few hours of the extraction the conjunctiva heals down at the edges and a closed pouch is formed. Under this complete covering the scleral wound remains open for several days: Dr. Lundsgaard will tell you that it may be open for five days. On the third or fourth day there is a tendency to hæmorrhage, and this blood may run into the anterior chamber, especially if the patient gets up. It may take weeks to absorb and can cause irritation.

The open wound invites prolapse, and in the case of the simple operation I have had several nasty extrusions of the iris. Prolapse is frequent after the simple method, but if we add a small peripheral iridectomy it is a very

rare complication. In this matter I am absolutely opposed to Col. Smith. I have never had a prolapse when I have nicked the iris at the periphery. I do not think that a complete covering of the wound with conjunctiva affords much, if any, protection against sepsis. In my last 100 extractions I have had the good fortune to lose but one from irido-cyclitis and this was a bridge operation. The bridge operation is that of election for a squeezer. When the section is completed the patient can press hard and yet the wound does not gape, and I have had no loss of vitreous in the twenty-five bridge operations that I have done.

A consideration of this operation guides us in our choice of incision : shall we make a corneal or a scleral incision covered by a long flap, or shall we reduce our flap to a mere frill? After a series of broad bridges I felt a revulsion against any conjunctival covering, and tried a few pure corneal operations. I am fairly certain that these latter have not healed so smoothly as those with a conjunctival flap. In some there has been an appearance as though the edges were over-riding somewhat, and in most of them for some time there was a fine trough in the line of incision. The medium course seems the best, not purely corneal, not a long broad flap, but a mere frill of conjunctiva. I am well aware that often, aim as we may, our actual incision is chosen for us.

I like preliminary iridectomy, and I am glad that Sir Anderson Critchett has given it his approval.

Mr. C. Killick

said he had done 1500 to 2000 extractions, but as a result of reading Terrien's recent book on the *Surgery of the Eye*, he had adopted this method of sub-conjunctival extraction which Mr. Harrison Butler did not like. The conjunctival bridge should be one-third of the corneal incision in breadth and prolonged backwards about as far as the insertion of the superior rectus. It was not a new opera-

tion, but was practised by Desmarres, and it will be found described and illustrated in the second edition of his work, *Les Maladies des Yeux*, published in 1856. Others took up the operation, but it had since fallen into disuse or disfavour. The speaker had done it nearly forty times. Sir Anderson Critchett had just said one must observe eclecticism in operations, for cataract; but for the ordinary type of senile cataract this was an excellent operation, and he would leave it to Mr. Cridland to describe its advantages. He would like to ask why the operation had been performed and then abandoned. Probably, he suggested, because it was a little more difficult than the ordinary straight-away operation. It would be found that there was some difficulty in getting the lens to present. He had found it difficult in his first few cases. If, however, the operator will add a millimetre or so to his corneal section the lens will readily escape, and he had not personally experienced the disadvantages that Mr. Harrison Butler mentioned. He had only done two simple extractions so far but both were successful. In the others he had done a very small iridectomy to the outer or inner side of the bridge—a far smaller iridectomy than he had previously performed. He hoped soon to be able to report a larger series of cases and to give detailed results. So far as he had at present gone he was highly satisfied with the operation.

Mr. Bernard Cridland

said that this had been rather a pet operation of his during the last few months. He had had forty-three cases, and he had not practised that eclecticism Sir Anderson Critchett advocated; he had done every one by this method, and without exercising any selection. He at first made his flap broad, but as time went on he found this breadth of flap unsatisfactory, and therefore he tried with a successively smaller and smaller flap, sometimes by accident, but mostly with intention; and now he did not

mind how narrow the flap was so long as it was as wide as a Smith's knife. The incision should be a little less than 180°. One lifted the conjunctiva on to the blade of the knife, and, with a turn of the wrist, slipped the knife right up beneath the conjunctiva, and it practically did the necessary cutting of itself. Of the 43 cases mentioned, 40 were simple extractions, 3 with iridectomy, 1 of them a little peripheral one. He had had 1 prolapsed iris, which was excised the following day, and the final result was good. Three were elderly diabetic women, with a rheumatic diathesis. In them he expected iritis on the ninth or tenth day, and it came; in one the other eye had been removed already, for the same reason. All the cases which had iritis did well.

Mr. Harrison Butler mentioned hæmorrhage following this operation. Two of the speaker's cases had had hyphæma, but that was due to trauma inflicted by the patients themselves putting their fingers into their eyes. He covered only one eye, and he allowed patients up 40 hours after the operation, but kept the eye covered 6 or 7 days. The average stay in hospital was 14 days; for cases which ran the routine course the time was 12 days; the average was extended by the cases of iritis.

He was charmed with the operation. Sir Anderson Critchett said operators must be eclectic, and it was right to choose one's cases for the operation. As Mr. Butler said, the lens should be left to choose its own outward route. Some iron-workers had very strong lids and could do almost anything with them, and but for the flap the operation would in them soon be spoilt. One could see the "squeezers" trying to do their worst, but without avail. In this series of 43 cases he had had no sepsis and no loss of vitreous.

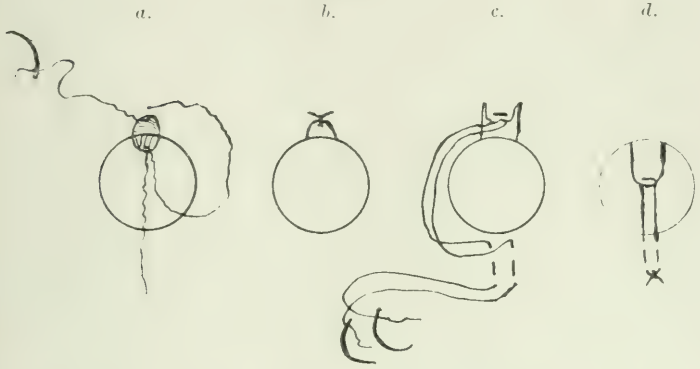
Mr. Maddox

said he was very interested in this operation with a conjunctival tether. He did many cases by it, but lately

he had rather abandoned it. He still preferred it, however, for eyes which were very prominent, in which there was a large extent of conjunctiva above. Deep-set eyes were not so dangerous. For these prominent eyes he used a flap of much the same breadth as Desmarres. In getting out the lens he found it a great help to seize the flap with blunt forceps and draw it down; that left a good space for the lens to come out.

Another way was Pansier's, namely after the knife had

FIG. 8.



come nearly to the top, to transfer the fixation forceps to the conjunctiva above the limbus, grasp it, and cut with a knife underneath the forceps; then one was ready, after again depressing the knife for capsulotomy, to express the lens without delay.

He had experimented a good deal with sutures for cataract extraction, trying eight or nine different methods, and he had selected three of them. A very good one was the following; it might be called a "pulley suture." He threw down a small 2 mm. flap, preferably one cut with a knife, and almost into the limbus (Fig. 8, *a*). Then he took the finest possible silk, passed it through this flap, and made a knot, he passed the needle round and through the upper edge of the conjunctival wound and then allowed

the needle to lie on lint on the forehead. The patient could be told that was the first stage of the operation ; and a pause, with speculum removed, would enhearten him to go through with the subsequent ones. He then passed the knife in the usual way, making a good large incision ; it was better too large than too small. The knife was brought out as shown, and the operation was then continued in the usual way. When the operation had been completed, the thread attached to the needle on the forehead would act like a pulley ; the flap was pulled up and the eye was drawn down at the same time, so closing the incision that vitreous could not escape. The eye could be pulled down sufficiently to make it easy to restore the iris. The suture was then tried (Fig. 8, *b*).

Another method was to cut a square flap upwards, pass a suture through, and let it retract upward out of one's way, as in Fig. 8, *c*. Needles were run through it, and then through the conjunctiva over the inferior rectus. The operation was done in the usual way and the suture tied afterwards. He did not like the second thread device so well as the first. The cornea tolerated sutures if they were properly chosen and were anointed with stiff ointment. Nothing could happen after suturing the flap in this operation even if the patient had an attack of coughing, or vomiting. There is quick healing, because the lips of the incision are pinched together ; and for the same reason there could be no prolapse. With a limbal incision there is often to be seen a slight ditch, filled with transparent matter, but that was avoided with this conjunctival cover, which, if placed properly and symmetrically, expedites healing.

Mr. Johnson Taylor

agreed with Sir Anderson Critchett that eclecticism was necessary. Apparently Sir Anderson objected to the simple method, and alluded to the difficulty of getting rid of soft matter. But it was a simple matter to get rid of

this material by washing out with sterilised salt solution, and the same process washed the iris back into position. He, the speaker, always tried to do a simple extraction. In his earlier days he had done a preliminary iridectomy, but not now, and he asked why two bites should be made at the cherry. In two or three cases he had known suppuration follow preliminary iridectomy. With the combined method, every now and then one got the capsule fixed up in the wound, with the occurrence of secondary glaucoma, which it was very difficult to deal with. It was clear from Sir George Berry's admirable book on practical ophthalmology that he inclined to the simple method, for he pointed out *inter alia* that one was less likely to get iritis if one did a simple operation than a combined one. He (Mr. Taylor) had noticed the same thing in the case of Elliot's trephining, for in the latter in his earlier cases he had every now and then failed to get the iris. When the iris was left intact he did not get iritis, but a little occurred when he got the iris out.

Why run two risks—by doing preliminary iridectomy—when one sufficed? And the patients in many cases would expect to be cured after the first operation however clearly you tried to explain the plan of campaign to them. He had recently devised double fixation forceps, and he did the whole thing in two or three strokes. He fixed the eye with the forceps, and did not leave go during the whole operation. He used to do capsulotomy with the point of the knife when making the section, but now and then a little aqueous escaped during this procedure, causing the iris to fall over the edge of the knife, which, with the shallowness of the anterior chamber, made it difficult or impossible to complete the section as intended, so he had gone back to the old fashioned capsulotomy.

He had seen one or two accomplished operators get into the same difficulty when attempting capsulotomy with the point of the knife as it passed across the anterior chamber.

Mr. Coulter

said sometimes it was two or three operations which were done, and patients did not like even two.

Mr. A. H. H. Sinclair

said that as some speakers had referred to difficulties occasioned by squeezing of the eyelids during the operation of extraction, he would like to emphasise the value of the injection of 2 per cent. novocaine, or better, a mixture of novocaine, cocaine and adrenalin, prior to extraction.

The subcutaneous injection of about 5 c.c. of this mixture is made into the temple near the external canthus. The needle is directed towards the eyelids, so that they become partially infiltrated. In this way the branches of the facial nerve passing to the orbicularis are temporarily paralysed and the lids at the same time become insensitive to the speculum.

In a few minutes after the injection the patient is unable to keep the eyelids completely closed and is quite incapable of squeezing them during the operation.

As the closure of the lids is imperfect the lids should be gently closed with the finger and a dressing of lint covered with vaseline placed over the eye. A suitable piece of cotton-wool and a perforated aluminium shield attached by strips of plaster complete the dressing.

He had also seen this injection used in Barcelona in March, 1921, but had employed it in Edinburgh during the previous winter.

He considered that the introduction of this method of ensuring quiescence of the orbicularis had proved the greatest advance of recent times in the technique of the operation for cataract.

3. *A note on the comparative values of artificial illuminants.*

By C. G. Russ Wood.

It is unnecessary to waste any time over the details of the ocular symptoms caused by the irritating rays in certain parts of the spectrum; these have been known for a considerable time. Briefly the violet end of the spectrum and, in greater degree, the ultra-violet are known to cause ocular troubles of more or less degree. They are chiefly due to variation in the retinal adaptation and irritation of the fifth nerve-endings in the cornea and conjunctiva. In artificial illuminants probably the latter effects preponderate, because it is known that the crystalline lens absorbs most of the violet rays and so forms an efficient natural protection. A clear glass globe or chimney has practically the same effect when surrounding a light.

If we consider the three stages of illumination as the rushlight, the bat's-wing gas light and the metal-filament electric bulb, it is evident that as methods of illumination have evolved, more blue and violet rays have been introduced into our domestic illuminants. Now light, when used for illuminating purposes, is obviously only utilised from the visible spectrum, and as the eye is most sensitive to that portion between the lines C in the red and E in the green, it is obviously advantageous, other things being equal, to utilise this part mostly on account of its superior defining power. The relative luminosity, according to Bell, of the various parts of the spectrum are roughly as follows: Crimson, 3 per cent.; scarlet, 20 per cent.; orange yellow, 98 per cent.; deep green, 50 per cent.; blue green, 7 per cent.; violet, 0·6 per cent.

As regards the quantity of light, visual perception is effective for ordinary purposes within a rather wide range of illumination brightness. According to the researches of Uhtoff, Koenig and Brodhun, with illumination below 3 metre candles shade perception is deficient, and that

practically light for illumination purposes when equal to about 25 metre candles is all that is necessary, and no advantage is obtained by increasing the illumination. Needless to say the distance the illuminant is from the object has to be taken into account, and as light varies as the square of its distance the source must be multiplied accordingly. Also many side issues have to be taken into account, such as the state of adaptation of the retina, the contraction of the pupil which reduces the visibility of objects in the visual field, methods of shading, the correct use of reflectors and flickering of the light.

We can now turn to the quality of illuminants, and in the first place should remember that daylight varies enormously in spectral quality, being altered by absorption in the atmosphere and selective reflections from the surrounding foliage, buildings, etc. For example, Nichols found on land the absorption spectra of chlorophyl which was absent on the sea.

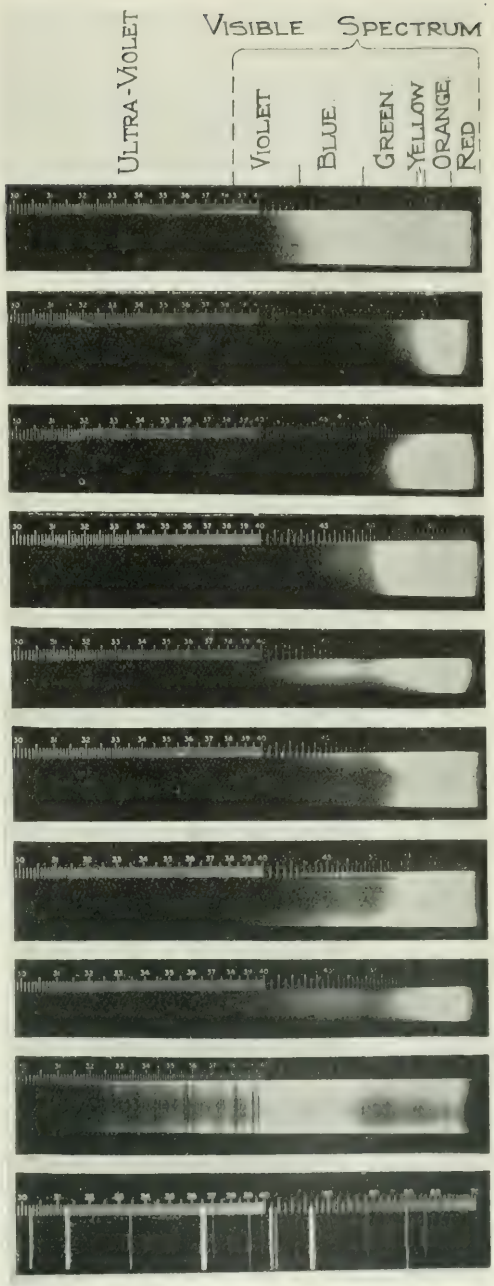
The fourth spectral photo shows one of the oldest forms of illuminant—a candle. Here is seen that red, orange, yellow and green of the spectrum are fully represented, with very faint traces of blue; the violet and ultra-violet are not present. There was no glass chimney around the light. The time of exposure was 6 minutes.

In the third is the spectrum of an oil lamp with a flat wick; here, contrary to what I expected to find, the spectrum is shorter and has less green, but red, orange and yellow are fully represented. This light again had no chimney. The time of exposure was 7 minutes.

The second photo shows the spectrum of the old-fashioned fish-tail coal gas burner. In this the red, orange and yellow are fully represented, but green only to some extent, roughly about one-half, while blue, violet and ultra-violet are entirely absent. There was no glass between the flame and the instrument. The time of exposure was 8 minutes.

The first photo is the spectrum of the incandescent gas mantle with no glass chimney, which shows full red,

Fig. 9.



The numbers indicate the wave-lengths of the light in millionths of a centimetre.

orange yellow, green and blue with violet represented roughly about one-half, but no evidence of ultra-violet rays. This exposure time was 1 minute 50 seconds.

In the spectrum of acetylene gas, the red is shortened, but orange, yellow and green are represented in full, with blue and violet partially. The exposure time was 4 minutes. No glass chimney used here also.

We now arrive at a different type of illuminant—the electric bulb, which is the mainstay of modern illumination. The carbon-filament form, which was the prototype, may appropriately be considered first. The filament is made by forcing a stiff solution of cellulose through a fine die. The resulting thread is carbonised, and after undergoing several processes the finished product is placed in the form of a loop inside a glass bulb which is open at both ends; the lower one is used to connect it with the electric wires, and the bulb is exhausted through the upper when it is finally sealed. In time these bulbs become black on the inner surface by minute particles of carbon deposited there from the filament. This decreases the useful life of the bulb considerably. The photo shows that red, orange, yellow and part of the green spectral areas are present with a slight amount of blue and less so of violet. Ultra-violet rays are absent. Exposure time 1 minute 50 seconds.

The next photo shows the spectrum of a Mazda metal-filament bulb, the exposure time being 2 minutes 15 seconds; red, orange yellow and green areas are fully represented with blue and violet partly, the former preponderating. No evidence of ultra-violet rays.

The next photo shows the spectrum of a metal-filament Osram gas-filled bulb. In this type the bulb is not exhausted but filled with an inert gas. This spectrum shows some shortening of the red end but full orange, yellow and green, with about the same blue and violet rays as in the exhausted metal-filament bulb. Exposure time 10 seconds.

I expected to find far more of the blue and violet

spectral areas in this illuminant on account of its intense whiteness and irritating qualities when looked at directly. The reason why this light has so much more brightness is because the filament is brought to a much higher temperature ; this intense heat can only be used on account of the inert gas—generally nitrogen—in the bulb. I am told that the glowing metal filament is at a temperature within 5 per cent. of its melting-point. Most probably the irritating nature of the light from a gas-filled electric bulb is due to its intrinsic brilliancy and not to violet rays.

Comparing these last three illuminants, for the same voltage, where the carbon lamp would give 1 candle-power, the vacuum metal-filament lamp gives 4 and the gas-filled bulb 8 candle-power. It is merely a question of the temperature at which the filament is used, and modern methods have enabled this to be very much increased. Of these three bulbs least value for money in illumination is given by the carbon and most value by the gas-filled variety, on account of the same amount of current producing so much more light.

The shadows on some of these spectra are probably due to the light sources being small in extent, and their images when thrown on the slit of the Hilger quartz spectrograph not being of uniform intensity throughout. The effect is not due to halation, but a difference in intensity from part to part of the flame. All the photos were taken on the Ilford process panchromatic plates.

In the illustration the lowest spectrum is that of the mercury vapour lamp, which consists of a tube filled with glowing mercury vapour. It gives practically a monochromatic light and plays tricks with colours, especially red, but is useful for seeing details in black and white ; Dr. Louis Bell found that it was superior to daylight for discriminating fine detail. This lamp is never used for ordinary domestic illumination and the spectrum is merely shown for comparison. You will observe it con-

tains a large number of the ultra-violet rays in spite of its being surrounded by a glass tube.

The daylight, not sunlight spectrum, is also merely shown for comparison; the shadow is due to the slit in the spectrograph being not quite perfect and closing more in the middle than at the ends. It will be seen that it shows a large amount of ultra-violet rays.

To sum up these results, we find all the spectral colours except the violet rays and merely traces of blue in the candle, oil-lamp and gas-flame spectra. This is what one would expect from the yellow and warm tints of these illuminants. In the incandescent gas mantle, acetylene, carbon filament, tungsten, vacuum and gas-filled bulbs are also found the warm-coloured rays with the addition of more of the blue and violet; this, again, is what we should expect from its much more white appearance. It is only in the last three bulbs that any glass was between the light and the spectroscope, so this may account for the absence of the ultra-violet, because glass absorbs a good deal of these rays. Therefore one is driven to the conclusion that the glare effects of some of these illuminants cannot be entirely caused by the irritating rays of the violet end of the spectrum, but probably in great measure to their intrinsic brilliancy. This conclusion agrees with the views expressed by Sir John Herbert Parsons. The general experience of most people is that yellow-green light is pleasant to work with. Luckiesh suggests another possible cause of the irritating nature of the more recent illuminants, namely, that they have a good proportion of the blue violet rays which cause chromatic aberration. It is difficult, as he points out, to focus blue light at a normal reading distance, and impossible to do this at the same time keeping the most luminous rays in focus.

This brings up the question, Which is the best form of artificial illuminant? The best reply is, I think, There isn't one. It all depends for what purpose the light is required. Personally I must confess to a sneaking regard

for a duplex oil table lamp, well shaded, for reading or even writing; but one must admit it has many disadvantages, such as the heat it gives out and the discoloration of the decorations of the room which it causes. There is no doubt that the newer electric bulbs give an excellent steady light, and if they are used intelligently with correct shades, of which the best type is the holophane, which absorbs less light than any other, they can be made to give great satisfaction. If general room illumination is required then the indirect methods of lighting are most satisfactory, especially if the colour-scheme of decoration be suited to the colour of the reflected light required. At the same time such a subtractive method of using light is wasteful from an economic point of view, but probably it is what will eventually be carried out in the future.

In conclusion, the general opinion that the yellow lights are the best for home use seems borne out by these photos. The causes of ocular illuminant irritation cannot be stated with certainty, but ultra-spectral rays, intrinsic brilliancy and chromatic aberration probably all play their part.

References.

Bell.—*Art of Illumination.*
Luckiesh.—*Colour and its Applications.*
Young, Meredith.—*Light and Sight.*
Parsons, Sir John Herbert.—*Lancet*, January, 1910.

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4. *The incidence of squint at various ages.*

By T. Stewart Barrie, M.B., Ch.B., F.R.F.P.S.G.

Introduction.

The subject of this contribution is given in the billet as "The Spontaneous Cure of Strabismus," but as a sub-title

more explicit in the meaning I now prefer "The Incidence of Squint at Various Ages."

The subject of strabismus is one of perennial interest, and has been studied from many aspects. The present is the statistical aspect. It has been said that statistics can be made to show anything, but on this occasion I have no ulterior motive.

I must define what I mean by spontaneous cure of strabismus. It is simply that the convergence disappears, and is replaced by parallelism of the optical axes of the eyes with or without the use of glasses. In many instances which have come under my notice parallelism has been established under conditions which exclude the usefulness of optical aids as in corneal opacities of the deviating eye; the cure has therefore resulted from other causes, and though anatomical reasons have been adduced, I am convinced that in many instances the cure is attributable to the passing away of what for want of a better name I call quasi-hysterical contraction.

Again, the definition of spontaneous cure of strabismus convergens which is herein given makes no reference to the state of vision of the two eyes nor to the vexed question of binocular vision.

One of the first questions which a parent or guardian asks when it is suggested that an operation for squint should be performed upon his child is—"Will the child not grow out of the squint?" The answer to this question is always guarded, because "as a matter of fact in many instances the squint gradually passes off as age goes on" (Sym.). This is true even when glasses are not worn for the correction of any refractive error present—in fact an amblyopic eye which deviates inwards in childhood may be found to be quite straight in adolescence though it still remains amblyopic. One may quite properly deduce from the statements of fact that there is a natural tendency for the angle of a convergent squint to become steadily less with advancing years; further, that the angle may disappear altogether, leaving the optical axes parallel; and lastly, by

a continuation of this reduction beyond parallelism, the eyes may become divergent. This change from a convergent squint in childhood to a divergent squint at the age of 21 has been noted by the writer in two instances, while the divergence of the optical axes which sometimes occurs in adult life following a tenotomy of the internal rectus muscle in early youth is to be explained by the same principle, and not to be rashly ascribed to faulty technique on the part of the ophthalmic surgeon concerned.

The evidence of the spontaneous decrease in the degree of strabismus is to be found in the case-books of every ophthalmic surgeon, but for statistical purposes it is valueless, and is vitiated by the fact that operative measures have been invoked in a number of instances. The best evidence is to be found in the reports of the examination of school-children at various ages, which within the last ten years have become of great importance. Unfortunately the condition of the eyes is not always reported by medical officers, but through the courtesy of Dr. C. C. Finlator, Medical Officer for the Clackmannan Education Authority, I have had access to his annual reports for the period 1911 to 1920, in which reports the condition of the eyes has been consistently given, and all cases of obvious squint have been noted. "Each scholar is examined as a matter of routine four times during his or her school life—that is to say, when first going to school, when seven years of age, when ten years of age, and lastly, when thirteen years of age, and therefore about to leave school. Each school is visited for routine inspections three times a year, and the medical officer is able to keep in closer touch with teachers and pupils than in the common method of doing all the routine work at one visit yearly" (report 1911—later).

When considering these reports a number of facts are to be borne in mind: (1) The age of each child is correct to within a period of four months; (2) strabismus is given in each report, but it is not stated whether it is of the convergent or divergent type, concomitant or paralytic.

This omission is of little importance for the present purpose and tends to reduce the number of cures; (3) the fact of the child wearing or not wearing glasses is not stated; (4) strabismus following the onset of phlyctenular keratitis or other conditions giving corneal opacities is not differentiated; (5) this area is a country area, and operative measures are not in favour with the people.

In the table which follows the numbers of boys and girls are approximately equal and have been added together.

Table extracted from the Annual Reports 1911–20, *giving the Incidence of Strabismus at Varying Ages in School-children.*

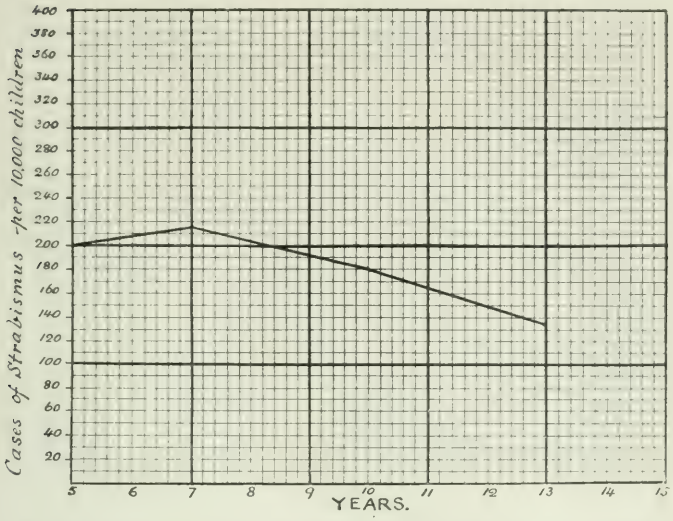
Year.	Age 5.		Age 7.		Age 10.		Age 13.	
	Children examined.	Cases of strabismus.	Children examined.	Cases of strabismus.	Children examined.	Cases of strabismus.	Children examined.	Cases of strabismus.
1911	632	16	549	7	350	4	582	10
1912	616	8	538	10	578	5	463	4
1913	647	18	573	13	611	12	597	9
1914	644	6	588	11	568	9	564	3
1915	556	15	548	11	540	9	531	8
1916	715	18	626	15	623	12	624	8
1917	573	14	580	17	613	17	510	12
1918	629	16	557	16	557	14	487	6
1919	559	5	536	9	541	7	491	9
1920	681	10	587	15	562	10	564	5
	6252	126	5682	124	5543	99	5413	74
Percentage of strabismus cases		2·01		2·18		1·79		1·37

These results can be expressed in terms which one can appreciate more readily by taking groups of 10,000 children at each age when the following is obtained:

5 years . . . 201 cases of strabismus,
7 ,, . . . 218 ,, ,,
10 ,, . . . 179 ,, ,,
13 ,, . . . 137 ,, ,,

or by constructing a graph in which the ordinates are ages and abscissæ are numbers per 10,000 :

Fig. 10.



Graph showing incidence of strabismus at ages from 5 to 13 years.

From these tables a number of deductions can be made viz. :

(1) That there is a small increase (9 per cent.) in the number of cases of strabismus between the years of 5 and 7.

(2) That there is a uniform decrease in the cases of strabismus from 7 to 13 years.

(3) That from the shape of the graph the decrease is likely to continue for some years after 13.

(4) That 32 per cent. of the cases of strabismus at 5 years are found to show no obvious strabismus at 13, and that 37 per cent. of those at 7 years are also found to have no obvious strabismus at 13 years.

5. *Persistent central scotomata following exposure of the eyes to direct sunlight during the solar eclipse on April* 8th, 1921.

By A. L. Whitehead (Leeds).

Four cases showing this lesion came under the observation of the writer.

(1) Miss E. D—, æt. 31 years, was first seen on April 16th, 1921.

No previous history of ocular trouble. On April 8th she watched the eclipse during the whole of its duration at intervals of a few minutes. Ever since there has been a sensation of a blind area in the centre of the field of vision, more marked in the right eye, with which the eclipse had been chiefly watched.

Vision in the right eye gave the first and last letters in the $\frac{6}{18}$ line, and in the left the similar letters in the $\frac{6}{6}$ line. Manifest hypermetropia +0·75 D. in each eye; no improvement with a correcting lens. There was a central positive scotoma in each eye up to about 5°. No fundus defect could be seen on this occasion.

She was seen again on January 25th, 1922. Vision in the right eye unaltered, in the left had improved to $\frac{6}{6}$ fully, but the central letters were not well defined. On this occasion a small yellowish atrophic spot could be seen on the outer side of each macula, that in the right eye being very definite.

(2) Mr. F. H—, æt. 29 years, was first seen on April 11th, 1921. He had used reading glasses for fifteen years, +1·0 D. sph. ⊃ +0·25 cyl. ax. 90°. He watched the eclipse at intervals and has had a partial central scotoma ever since. V.R. $\frac{6}{18}$ end letters only; V.L. $\frac{6}{9}$ end letters only. No correcting glass improved. No fundus changes could be seen. On June 1st, 1921; vision had improved to R. $\frac{6}{9}$, L. $\frac{6}{6}$, and he was no longer conscious of any blind area.

(3) Mrs. R—, æt. 45 years, was first seen on June 13th,

1921. She had not experienced any trouble with her eyes prior to the eclipse, which she had watched at intervals, using the left eye almost entirely.

V.R. $\frac{6}{6}$; V.L. $\frac{6}{12}$, with a small central scotoma involving single letters in this line. The right fundus appeared normal, in the left there was a small atrophic spot on the outer side of the macula.

In August, 1921, and again in April, 1922, the scotoma, vision and fundus were unaltered.

(4) Mrs. S—, æt. 37 years, was first seen on May 11th, 1921. No ocular defect prior to the date of the eclipse; she had watched the sun at intervals and has had a bilateral central scotoma ever since, the left eye being more affected.

V.R. $\frac{6}{24}$; V.L. $\frac{6}{24}$; refraction normal. There was some physiological accumulation of pigment round the left disc, and at each macula a small yellowish atrophic spot. In September, 1921, vision and fundus were unaltered.

In none of these cases was any smoked glass or other protection used, and the observations were mostly made with one eye, the other being wholly or partially closed; in the case of Miss E. D— the whole process of the eclipse was watched at intervals of a few minutes, while Mr. F. H— made only five or six brief observations.

There was no pain, no photophobia or discomfort of any kind beyond the central dazzling scotoma.

The literature on these cases is scanty and affords very little help regarding prognosis.

Definite changes at the macula have been recorded in some cases, chiefly pigment proliferation with permanent defect in vision. The fundus lesion described in this series of cases does not seem to have been previously reported. Small atrophic spots of this character are seen in other cases, but are rarely present at the macula, and therefore any resulting defect in vision is unnoticed. Their presence always in identical positions—that is, just to the outer side of the macula in each case of persistent

scotoma—seems to be more than a coincidence and therefore justifies publication.

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6. *Trachoma of the lacrymal sac.*

By A. F. MacCallan, C.B.E., and M. Sobhy (Egypt).

The short note which follows on the subject of trachoma of the lacrymal sac should be treated as a preliminary communication only. Unfortunately the time at my disposal for study is limited by the demands made upon me by the administrative business of twenty ophthalmic hospitals, by biennial post-graduate courses of ophthalmology, and by the clinical work of two hospitals in the outskirts of Cairo, at each of which there are between 300 and 400 patients a day. However, having been engaged in the organisation of these hospitals now for nearly twenty years and my pupils being apt at details of administration, I hope that I shall soon be able to hand over the active direction of the hospitals to one of them, and devote myself to clinical research, the practice of ophthalmic surgery and the teaching of ophthalmology.

My attention has been intermittently drawn to chronic dacryocystitis in Egypt on account of its frequency. Thus it was found in:

Year.	Cases of dacryocystitis.	Total cases seen.
1921	719	113,000
1920	780	94,000
1919	592	76,000
1918	635	82,000

It is probable that there were a good many more cases in which the dacryocystitis, although noted on the clinical record, was not the main reason for the patient seeking treatment at the hospital, and so, not appearing as the diagnosis of the case, was not noted in the statistics.

Extremely poor results have attended treatment by syringing the sacs, while removal of the sacs has usually been a satisfactory proceeding provided that the patient has not got serious disease of the nasal mucous membrane. Those operations which depend on the reopening of a communicating channel with the nose, belonging in my opinion to the domain of the rhinologist, I have no experience of. It is my belief that with, at any rate, a large number of sacs, the mucous membrane of which is trachomatous, the only treatment of any practical value is removal of the sac. With a trachomatous sac an opening into the nose is very liable to become blocked either by hypertrophy or cicatrisation of the mucous membrane.

Trachoma of the lacrymal sac is a well-known complication of trachoma; more than this, trachomatous dacryocystitis as a primary disease has been described by Hoffmann. I can put no credence in trachomatous dacryocystitis as a primary condition, knowing as I do the occasional difficulty of being certain as to whether trachoma of the conjunctiva is or is not present, when there are no obvious follicles, no cicatrisation, none of the peripheral pits described by Herbert, and only the faintest vestiges of corneal pannus, as determined after examination of the cornea with a good loupe, my suggestion being that in these cases of reported primary dacryocystitis there is undetected trachoma. I may mention that it has taken me a long experience in a trachomatous country to make me aware that in a few cases a complete certainty of previous trachoma of the conjunctiva is almost impossible.

In cases of trachomatous dacryocystitis where the conjunctiva has become partially cicatrised or cured (MacCallan's stage III), there is a danger of reinfection of the conjunctiva from active disease in the lacrymal sac.

The possibility of the trachomatous infection making its way into the sac *viâ* the nasal mucous membrane has been considered, but there are no grounds for the supposition.

Dr. Sobhy has prepared microscopical specimens from the lacrymal sacs of eighteen patients whose sacs were excised on account of dacryocystitis. I have here four of these in which trachomatous follicles are seen, and a fifth case in which the sac is the seat of hyaloid degeneration (Path. Reg. Nos. 372 [1921], 85, 87, 184 [1922]).

The follicles are seen to be made up of two zones, one a clear central zone and the other a peripheral darker zone. In the central zone large mononuclear leucocytes predominate with more epithelioid cells. In the peripheral zone small lymphocytes are predominating.

Trachoma of the lacrymal sac is referred to by Elliot (1) in his text-book and by Axenfeld in his book on the *Bacteriology of the Eye.*

Meyerhof was kind enough to give me an abstract from the important article by Cirincione (2). I have studied the diffuse but important communication by Kuhnt (3) on the condition of the lacrymal apparatus. Other references have been given to me by Meyerhof and appear in Axenfeld's book (4).

I hope at the next meeting of this Society that I shall be able to give the results of the examination of 100 sacs. The difficulty I foresee is to obtain a few sacs from non-trachomatous subjects, and for this I must ask my colleagues in England to help me by sending to me in any well-known fixative the results of some of their operations.

References.

(1) Elliot.—*Tropical Ophthalmology,* 1920.

(2) Cirincione.—"Tracoma dei canalicoli lacrimali," *Annali di Oftalmologia,* 1890, xix, p. 362.

(3) Kuhnt.—"Die Therapie du Conjunctivitis granulosa," *Klinisches Jahrbuch,* 1897, vi, p. 439.

(4) Axenfeld.—*Bacteriology of the Eye,* Macnab's translation, pp. 277, 284.

7. *Dry sterilisation of instruments.*

By C. F. Bentzen.

The reason why I have asked for the honour of speaking here to-day is that I read an article by Dr. W. H. Simpson in *The British Journal of Ophthalmology*, November, 1921, about "safety in ophthalmic operations." Dr. Simpson tells us there that the ordinary way of sterilising ophthalmic instruments in England is by boiling them for five to ten minutes in distilled water to which a little sodium carbonate is added.

This method is safe, of course, and when I am in a hurry and have not arranged matters beforehand, I use it myself, with a slight modification, indicated by my Danish colleague, Dr. Claudius, who makes the boiling less damaging to the instruments by adding 1 per cent. of sugar to the sodium carbonate.

But for the last fifteen years I have sterilised my instruments by dry heat, heating them to 150° C. (302° F.) for thirty minutes in a gas-stove. This method is just as safe as boiling.

I have tested its reliability on several occasions. The last experiment was made with eight cultures of staphylococcus and ten cultures of soil spore. The result was that all the staphylococcus cultures and seven out of the ten soil spore cultures were sterilised. Considering that boiling does not kill soil spores the result must be regarded as a good one. The great superiority of dry sterilisation over boiling is that it does not spoil the edge of the cutting instruments. If you boil your cataract knives you must have them sharpened after having used them once or twice, and not even the first time you use them can you be sure that the edge is good.

By using dry heat you can operate with the same knife many times without having it sharpened. A few months ago I used an old cataract knife eleven times before I thought it safest to have it sharpened again.

By not having your instruments sharpened so often they will last longer and you will save money in two ways.

After dry sterilisation the instruments are dry and clean, free from any deposits that may stick to them if they have been boiled. If the instruments are properly covered and kept in a dry place they will keep sterile for any time and are always ready for use. In that way dry sterilisation will save time for the operator.

Before the sterilisation I have my instruments arranged in a metal case with overlapping cover. The case is wrapped up in two pieces of paper, according to Prof. Bloch's method. When the instruments are wanted any person can remove the outer piece of paper and hand the sterile contents to the surgeon. The lid of the case can be used as a second tray. The two racks on which the instruments rest are not parallel, to allow for the different length of the instruments. For single instruments, knives, needles, etc., Dr. Lundsgaard has constructed a wire frame, which is kept in an ordinary culture-glass.

The stove I have used these fifteen years is an asbestos-covered gas stove, with double walls and divided into three rooms by two asbestos-covered shelves. The inner measurement is about one cubic foot; the price is about £8. A double cock is inserted in the india-rubber gas-tube for finer regulation of the flame. The temperature inside the stove is not the same everywhere. It can be up to 50° C. (122° F.) warmer at the bottom, where the burner is, than under the ceiling. Therefore it is only possible to use the top room for the instruments, which must be placed at the same level as the control thermometer.

It takes about forty-five minutes before the temperature in the stove rises to 150° C. (302° F.). The stove must be watched during sterilisation to prevent the temperature sinking, whereby sterilisation is insufficient, or rising too high, the instruments getting spoiled. At 180° C. (356° F.) they turn yellow, but can still be used, but at 200° C. (392° F.) they turn blue and are completely spoiled.

Self-regulating electric stoves give less labour to the *personnel*, but they cost double the price, and the one sample I have tried was not able to produce a temperature of 150° C. (302° F.).

If any of the surgeons present will give dry sterilisation a trial I shall be very glad, and I am sure that you will be just as pleased with it as I have been.

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8. *Notes on light-sense.*

By A. S. Percival.

I wish to make a correction in the paper on "Light-Sense" that I read here two years ago. Further experience has confirmed my view of the importance of the subject, and has shown me that in one point I made a grievous blunder. I said that L.M. is most affected in diseases of the retina and choroid and in glaucoma; while L.D. suffers chiefly in diseases of the optic nerve. I now find that in superficial retinitis (the nerve-fibre layer) L.D. suffers very seriously. I have long noticed that toxic amblyopia was characterised by a striking failure in L.D., and as I suppose that most of us consider the site of the lesion to be the ganglionic layer of the retina, this observation is quite in line with my present finding that L.D. suffers when the retinal nerve-fibre layer is at fault. I owe to Dr. Traquair this neat way of expressing the distinction: that in L.M. failures the receiving apparatus (the visual purple and therefore the choroid and bacillary layer) while in L.D. failures the conducting apparatus is at fault.

It will be found that if dark glasses are worn, after a few minutes the L.M. and L.D. tests will give exactly the same results as they do with the unshielded eye. Hence the observations made on eyes with turbid media or cataract with these light-sense discs can be relied on when no details can be seen with the ophthalmoscope. Last year a patient came to me with advanced cataract in

her left eye, a less mature cataract in her right eye. Her left eye gave L.D. as 50, but her L.M. was less than 12·5. I concluded that there must be a macular hæmorrhage, and refused to operate on that eye, and warned her that such an operation would not be successful. However, she subsequently was persuaded to have the operation done, with the usual disappointing result.

Some have complained of the difficulty in getting the patient to understand what they are required to see when rotating the discs. Let us consider the L.M. disc with the smaller sectors as an example. I now always ask: "How many perfectly black rings do you see"?

If the patient says "Two," it is clear that he must see three grey rings distinguished from the two black rings, and his L.M. is 200. If he sees only one black ring, his L.M. is 100. If it does not appear quite uniform in tint, his L.M. is 50. If uniform in tint, his L.M. is less than 50.

Similarly with the L.D. discs, ask—"How many perfectly white rings do you see," and draw your conclusions as before.

Copies of the following Notes on Light-Sense were handed round, and Mr. Percival mentioned that the discs could be obtained from Messrs. Robson & Co., 46, Dean Street, Newcastle-upon-Tyne.

Notes on Light-Sense.

Light sense near the threshold is an independent function of vision almost certainly due to the visual purple and the retinal rods, but not the cones; it is far more primitive than form sense. Children with C.C.S. and amblyopia of the squinting eye have usually a much better light sense in their amblyopic eye: this extraordinarily good light sense may be lost in later life.

It will be found that in diseases of the choroid, or of the deeper layers of the retina, the sense for light minimum (L.M.) is most at fault; whereas in diseases of the conducting apparatus (*e.g.* the optic nerve and even the

retinal nerve fibrils) the sense for light difference (L.D.) suffers most (this may easily be remembered by associating L.D. with o. d.).

Thus L.D. fails in optic neuritis, retrobulbar neuritis, n toxic amblyopia, and in superficial retinitis.

The L.M. fails in deep retinitis, choroiditis, macular hæmorrhage, etc., and also in night-blindness even when no change in the fundus is seen, as in coal-miners, which I attribute to over-activity of the visual purple, and hence an increase of the endogenous stimulus. (*Trans. Ophth. Soc.*, vol. xl, p. 321.)

Hazy media and early cataract do not interfere with the reliability of these statements, as may be confirmed by trial on viewing the discs through dark-tinted glasses.

I believe that the earliest sign of glaucoma is a failure of the L.M. sense due to injurious pressure on the sensitive bacillary layer, and that this is far more reliable than any single tonometric test. Later, as the nerve fibrils become affected and when the optic disc becomes cupped there will be a failure of the L.D. sense.

On this, and indeed on all my observations, it is most important to have the confirmation or the refutation of others.

The tests with the rotating discs can be carried out in ordinary daylight.

L.M. discs.—Two black discs on each of which are three white sectors of different sizes; when one of these is rotated three lighter rings are seen, of which the inner is the lightest. On the disc with the larger sectors, the inner ring denotes a L.M. of $\frac{1}{1\cdot\frac{1}{2}\cdot\frac{1}{5}}$, the intermediate ring $\frac{1}{\frac{1}{2}\frac{1}{5}}$, the outer $\frac{1}{\frac{1}{5}\frac{1}{0}}$, which it is more convenient to denote by 12·5, 25, and 50, as ability to see the outer ring denotes a better light minimum sense than that which can only see the inner ring. Similarly the disc with the smaller sectors denotes L.M. of values 50, 100 and 200.

L.D. discs.—These are white discs with black sectors of different sizes, and when rotated indicate the L.D. sense for values 12·5, 25, 50 and 50, 100, 200.

It will be found that few can distinguish the faint grey ring that corresponds to L.D. = 200, so I assume that the standard for L.D. is 100, while that for L.M. is 200.

It will be noticed that a light sense of either kind, of value 50, is given twice; this is often useful in forming an opinion of the value of the statements of the patient.

9. *A neglected factor in the ætiology of miners' nystagmus.*

By A. S. Percival.

Those who have had practical experience of this disease will be as dissatisfied as I am with the general tenor of the first report of the Miners' Nystagmus Committee. The evidence given for their decisions is extraordinarily inconclusive.

It is the fashion for the moment to attribute the disease *wholly* to the deficiency of light. How, then, do they account for Snell's case of the disease in a ceiling paperer? I have had one or two cases where there was no deficiency of light. Such cases are not mentioned.

On p. 29 we are told that "there does not appear to be any definite connection between the thickness of the seams and the incidence rate. In support of this statement, which is in direct opposition to Snell's painstaking work on the subject, they give their Table VII, "showing thickness of seam in which 1613 coal-face men worked." However, on looking at the table we find that the number of *cases of nystagmus* is 1613, and that only one case of nystagmus occurred where the thickness of the seam was less than 2 feet, while 650 cases were found where the thickness of seam was 6 feet and over.

Such a table is absolutely useless, unless the number of men employed at each thickness of seam is given. Obviously more men are employed in the thick seams than in those less than 2 feet. There might be only two men employed in the seam less than 2 feet, and 40,000 in the seams 6 feet and over.

On p. 40 there is a very curious example of their reasoning :

"If lights amounting to 100 candle-power are placed in a room having walls, floor and ceiling of reflecting power 80 per cent., one-fifth of the light will be absorbed and four-fifths reflected."

Certainly.

"This reflected light is for all intents and purposes a new light of 80 candle-power. The total effective light in the room is therefore the same as if the walls and ceiling had been black, and 400 lamps, of 1 candle-power each, had been spread evenly over the walls, in addition to the original 100 candle-power."

But it is to the amazing ignorance of first physiological principles that I wish to draw attention. The miner cares not in the least for the total effective light in the pit ; what he does care about is the light on the coal-face he is hewing.

Everyone knows that in a fairly dark room contrast of illumination is of supreme importance ; one will see an object illumined by a $\frac{1}{2}$-foot candle when the rest of the room is lighted by a $\frac{1}{10}$-foot candle far better than if the whole room has an illumination of $\frac{1}{2}$-foot candle.

I have no wish to open a discussion on the general ætiology of miners' nystagmus in the absence of the special committee appointed to investigate its causation.

It is a common observation that those who make the most dogmatic assertions about a subject are those who know least about it.

What can be clearer than the wave theory of light which was dogmatically asserted thirty years ago ? It is now known that the whole theory must be recast in accordance with our present view of matter—that of whirling electrons—and to explain the conflict between the observations that have been made. The Michelson-Morley experiment definitely suggests that the ether is dragged by the moving earth, and the phenomenon of stellar aberration as definitely suggests that the ether is stationary. However, I am not going to talk about Einstein ; but to illustrate the difficulty of forming the new theory I may point out that although the fourth

volume of Poynting and Thomson's standard text-book on physics was published in 1914, we are still eagerly waiting for the fifth volume on light.

It is time to throw all our old views of nystagmus into the melting-pot, and to take an entirely new view of the whole subject, as has been found necessary in physics.

I may very reasonably be asked for my credentials as to my experience of the matter. At the Newcastle Eye Infirmary I see about 100 new cases of real or suspected miners' nystagmus a year, and I have been required for the last year to report on 140 cases of nystagmus as ophthalmic referee to the two Northern Circuits (Nos. 1 and 2).

In the North about 5 per cent. of all those working in the pits have been certified as suffering from miners' nystagmus, and, owing to the Workmen's Compensation Act, it is we, the coal buyers, not the colliery owners, nor the insurance companies, who finally pay for the compensation in the enormously increased price of coal. Many of our industries have been paralysed by it, and our export trade has suffered enormously.

We have followed each other like sheep in the same old path too long.

What are the outstanding facts about the disease? Surely that anyone who has once had miners' nystagmus, and after a rest, followed eventually by surface work, shows no symptoms of it, will certainly have a recurrence if he returns to hewing. I have had four cases sent to me from the county court registrars on each of whom I had reported previously three or four times. They each acquired nystagmus, obtained compensation, lost all their symptoms, obtained work at another pit as hewers, again acquired nystagmus, again obtained compensation, got relief from their symptoms, again obtained work in another pit as hewers, and again obtained compensation from this third pit for nystagmus.

I submit that, say, 5 per cent. of all children born have a predisposition to the disease, and if they have not this predisposition they will never acquire it. If they have

this predisposition they have it for life. Unfortunately no means have yet been discovered for recognising the predisposed before the onset of the disease.

We are all familiar with cases of heterophoria, as shown by Maddox rods, which yet present no symptoms. I have about 6 ∇ of exophoria at reading distance; my colleague, Mr. Bennett, has about 3 ∇ of hyperphoria at 6 metres; we neither of us have any symptoms from this heterophoria. Indeed slight heterophoria is far more common than orthophoria, and we are asking for trouble if we attempt to correct it in the absence of symptoms of discomfort. In his lecture here last year Dr. Maddox talked about the central apparatus that directs the two eyes to the same object, and it is apparently to the extraordinary efficiency of this nervous structure that most heterophorics escape untoward symptoms. But some are not so fortunate, and may be distressed by even ½ ∇ of hyperphoria. Their fusion centre is not so efficient, and one may say that they are predisposed for that reason to suffer from heterophoria. The predisposition to nystagmus I take it depends essentially upon a congenital inadequacy of a similar centre which perhaps may be called the fixation centre.

If my contention be justified, the Workmen's Compensation Act is fundamentally unjust. Practically it means that the patient receives whole or part compensation for the rest of his life, for there are very few workmen who are wise enough to seek other employment. It is bad for the workman pecuniarily, for whatever his ability in other ways he can never make much money, even with part compensation, while doing light jobs at bank; and it is demoralising to the praiseworthy principle that underlies the British workman's love of independence and hatred of charity.

It is obvious that the unfortunate pitman who acquires miners' nystagmus must be given compensation for a limited period, say, twelve months or so, while recovering from his symptoms, and to give him time to obtain other

employment, but that he should never be allowed to obtain underground work again.

This is not introducing a new principle, for under Section 8 (1) (*b*), one reads: "If it is proved that the workman has at the time of entering the employment, wilfully and falsely represented himself in writing as not having previously suffered from the disease, compensation shall not be payable."

Finally, there is the great difficulty about the "odd lot" question.

Let me refer to the case of Kear *v.* Shelton Iron, Steel & Coal Company, heard in the Court of Appeal, May 2nd, 1921.

A miner with miners' nystagmus was given light work on the surface. He did this work for about a year, and received the difference between his then earnings and his earnings before the incapacity, as compensation. He was then dismissed, with many other men, in no way incapacitated, on account of slackness of trade.

The county court judge found that he was capable of "doing quite a number of labouring jobs, but not of *any* labouring job that might be open," and that this was the result of nystagmus. He thereupon awarded compensation for total incapacity.

The appeal failed—the learned judge had in effect found that the man had not the capacity of an ordinary labourer by reason of nystagmus, and was therefore an "odd lot."

After this decision, from some pits I had a large increase of slight nystagmus cases which I signed up as able to do light work above ground. The colliery owners were overwhelmed with disabled soldiers to whom had been given all the light work available, and so were unable to provide any suitable employment to these men. I understand that many of them applied for work for which they were entirely unfitted, and then as a result of this decision applied for and obtained compensation for total incapacity as "odd lots."

Now my contention is that miners' nystagmus is not caused by hewing in the same sense as lead poisoning is caused by exposure in white lead works; work underground only reveals the disease, and shows that they are

unfit for that kind of work, just as I am totally unfit for the work of an R.A. Almost every other kind of work is open to them, and the interests of both workmen and employers would be best served by limiting the period during which the compensation could be claimed, and encouraging the workman to obtain another kind of employment. There should be no difficulty in young men obtaining other employment, but in the case of men over 40 or 50, the period during which compensation is obtainable should be increased.

10. *Upon changes in refraction.*

By T. Harrison Butler.

The object of this paper is not so much to announce new facts or theories, but to introduce a discussion upon a question which is of great importance to beginners in ophthalmic practice, and to stimulate research upon the subject. Such a research, owing to the slowness of the changes in adults, requires from fifteen to twenty years, and must be undertaken by those who are commencing work. If these start with a correct idea of the variations which may be expected, they will not overlook the facts as they present themselves, and will be able to interpret them correctly.

In general the refraction of an adult is stable, and changes, if at all, along well-recognised lines. In a minority alterations are found which may be regular and progressive, or irregular and oscillating.

There is no such thing as astigmatism with the rule in the strict sense of the word. It is usual in the young to find the convex cylinder vertical and the concave horizontal, but in the old the reverse is more common. Hence there must in many persons be a tendency for one type of astigmatism to change into the other. Examination of a large number of patients watched for over ten years shows that this is the case.

It is convenient to divide the cases into two groups. In the one we place children of school age; in the other adults. In other words we consider changes in the growing and in the fully-developed eye.

Changes during the School Age.

This is the more difficult investigation, because we are not always certain of the precise refraction. It is mostly determined by objective tests, and often the exact figures, which can be obtained only by subjective methods, are not at our command. There is an error—and it may occasionally be a large one—introduced by incomplete cycloplegia. Experience at the Coventry and the Warwick County School Clinics, extending over ten years and comprising the examination of over 1000 children a year, has shown that if the atropine be instilled by the mothers at home for three days, or at the clinic itself for an hour or more, cycloplegia may be incomplete. For exact research atropine must be used for three days and instilled by a competent person. Making due allowance for incomplete cycloplegia, it has been found that the refraction of school-children may undergo great changes during the school period—from 5 to 14 years of age.

Myopia.—The changes that take place in myopic children are well known, and there is nothing new to be said. There is a general tendency for myopia to increase at a uniform rate of about 1 D. a year, and some cases even during school life begin to show evidence of deterioration in function. In the majority of the children it has been possible to trace evidence of heredity; many of the mothers say with pride that the grandmother was able at 70 to do the smallest needlework without spectacles!

There is not the same tendency to advance in cases of simple myopic astigmatism, and even in compound myopic astigmatism the eye appears to be more stable than the pure myopic eye.

Hypermetropia.—Many children will be found to show a steady diminution in hypermetropia, and even to become

myopic. High-grade hypermetropia is not at all uncommon among children, but we do not find it so frequently in adults. It follows that there must be a striving towards emmetropia during growth. This change has been frequently observed at the Coventry School Clinic; hypermetropias of over 6 D. have become reduced to 4 D. and even less during a period of six years. Probably the majority of high hypermetropes have become moderate hypermetropes when adult life is reached. Others do not alter much. In many of these cases the reduction appears to be a mere growth change—a benign adjustment of the axial length to the dioptric strength of the lens system. In others it would appear to be of the same nature as the changes which we find in myopia. It is pathological, and some eyes may show myopic fundus changes, such as a crescent, although the refraction is still hypermetropic. It would be an interesting study to trace the fate of these eyes. The alteration is the same in both eyes, but in myopia one eye may get ahead of its fellow and catch it up later on.

Astigmatism.—Changes in the cylindrical element of the refraction are apt to be irregular in children, and are difficult to reduce to rule.

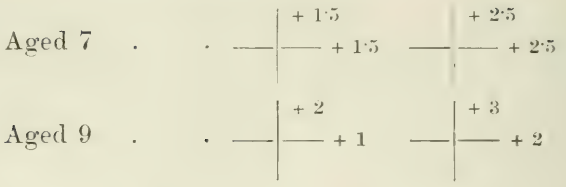
Alterations in angle are quite common, but in small children the inclination of the cylinder must be guessed from the retinoscopy, and there is scope for considerable error. Many of the older children will give valuable information respecting the correct angle, and even in such cases notable changes have been found after two or more years.

Alterations in the amount of the astigmatism are very common—in fact, almost the rule rather than the exception. They may be regular and progressive, or irregular.

As an example of the regular type we have—

Doris S— :

Aged 6 . . $\frac{+0.5}{+1}$ $\frac{+1}{+2}$



It will be noted that although the horizontal measurement remains nearly stationary, the vertical increased from 0·5 to 2·0. This is an instance of astigmatism with the so-called rule becoming astigmatism against the rule—a change which is very common in the adult eye after 45 years of age, but not so often seen in children.

M. C— is an example of a change in the reverse direction. The astigmatism with the rule has steadily increased from 1·5 to 4. Here the glasses ordered were as follows:

Aged 4 . . .	+ 3·5	+ 3·5
	+ 1·5 75	+ 1·5 ↓
Aged 10 . . .	+ 1·5	+ 1
	+ 3 75	+ 3 ↓
Aged 11 . . .	+ 0·75	0
	+ 3·5 75	+ 4 ↓

This is also an example of decrease in hypermetropia. The astigmatism had increased at the expense of the hypermetropia, in such wise that the sum of the sphere and cylinder remain nearly the same. A similar change is seen after a cataract extraction. The high astigmatism associated with low hypermetropia found during the first fortnight alters so that the cylinder diminishes and the sphere increases in such proportion that the sum of the two remains the same.

The building up of the final refraction as seen during the school age is an interesting study and one worthy of careful investigation.

Changes After School Age.

Here we are on surer ground; we can rely upon our private cases, which are investigated under the best conditions.

The changes found after school age are smaller in amount than those which take place during the growth of the eye, but they are far more regular, and, given adequate time, are easier to study and classify. If the work be done conscientiously there is no room for more than minimal errors.

Myopia.—Here again there is nothing new to relate. It has been seen that during school life myopia tends to a gradual but steadily progressing increase. During later life it may take one of three courses. It may steadily develop, and the eye may eventually become useless by reason of degenerative changes at the macula and elsewhere.

The second class develops up to, say, 4 D. and then halts. All seems well, but after a certain time, which may extend to several years, a secondary advance starts. This process may be repeated and may follow a debilitating disease such as influenza. The ultimate prognosis is far better than in the first class. Finally we have cases which reach a low degree of myopia, say up to 3 D. and then never alter.

Hypermetropia.—In the majority of persons hypermetropia appears to be stationary when the eye is fully grown, and no change takes place till late in life, when in some cases there is a senile increase in the hypermetropia. This is equal in both eyes and only exceptionally exceeds 1 D. This senile change may occasionally start at about 45 and then may rise to 1·5 D. It is suggested that the frequency of the senile increase in hypermetropia has been exaggerated in the text-books. It would appear that the tendency is for hypermetropia to diminish during the period of growth, then to remain stationary till 50 or 55, when in some cases it again increases.

Astigmatism.—Astigmatism may alter during adult life in two directions. The quantity may vary, and the angle may alter.

Alterations in the amount of astigmatism.—There is a tendency during life for the constant pressure of the lids to flatten the cornea in a horizontal direction in such wise that astigmatism with the rule tends to diminish, that an eye without astigmatism may develop it against the rule, and that astigmatism with the rule may become astigmatism against the rule. This is one of the methods by which this change takes place. The same transformation may be effected by rotation of the angle of the cylinder. These changes may be gradual, but may also occur late in life with comparative rapidity.

The following are examples:

Miss J—:

Aged 57 . .	0 / + 1·5 ↓	+ 0·5 / + 1 ↓
Aged 58 . .	0 / + 1·25 ↓	+ 0·5 / + 0·75 ↓
Aged 60 . .	0 / + 1 ↓	0 / + 0·5 ↓

This is a typical example of a regular diminution of astigmatism with the rule taking place after the age of 50.

Miss L—:

Aged 45 . .	+ 0·5 / + 0·75 120°	+ 0·75
Aged 51 . .	+ 0·75 / + 0·5 135	+ 1
Aged 62 . .	+ 1 / 0	+ 1·25

This is an example of diminution of the amount of astigmatism with the rule, swinging of the axis towards the horizontal, and increase of hypermetropia, all after the age of fifty.

The reverse change may take place, but it is quite uncommon.

The following is an extreme example :

Miss P— :

Aged 20 . $\begin{cases} \text{V.R.} = \frac{6}{3} \ \bar{c} + 0{\cdot}25 \downarrow \text{clearer.} \\ \text{V.L.} = \frac{6}{3} \ \bar{c} + 0{\cdot}25 \downarrow \text{clearer.} \end{cases}$

Aged 29 . $\begin{cases} \text{V.R.} = \frac{6}{12} \ \bar{c} - 1{\cdot}5 \ {}_{170^\circ} = \frac{6}{3} \text{ partly.} \\ \text{V.L.} = \frac{6}{9} \ \bar{c} - 1{\cdot}5 \ {}_{180^\circ} = \frac{6}{3} \text{ mostly.} \end{cases}$

This is one of those interesting cases with an acuity of $\frac{6}{3}$. They are very rare, and are apt to suffer from asthenopia if there is the slightest astigmatism. In this patient violet headache was present. Plus cylinders of 0·25 D. axis vertical were ordered and cured the headache completely for some years. Nine years later headache returned and diminished acuity was noted. It was now found that the astigmatism had become myopic and had increased to 1·5 D. This correction gave an acuity of $\frac{6}{3}$ mostly, and again cured the headache.

Alterations in the Angle of the Cylinder.

In general the angle of a cylinder remains constant but changes are frequent. The common tendency is a steadily progressing swing of a convex cylinder from the vertical towards the horizontal. In a few cases the reverse swing may be noted, and in others again there may be an oscillation of the angle through a small angle.

The horizontal tendency in the swing is one of the ways by which astigmatism "with" turns "against" the rule. I have so far not traced the cylinder right round from 90 to 180 but I have no doubt that the full swing takes place.

The following are examples of swing :

Mrs. F— :

Aged 41 . . $\frac{-0{\cdot}5}{+2 \ {}_{115^\circ}}$

Aged 44 . . $\frac{-0{\cdot}5}{+2 \ {}_{125^\circ}}$

Aged 50 . . $\frac{-0{\cdot}5}{+2 \ {}_{130}}$

Miss O—:

Aged 51 . .	-4	-3	
	$-0{\cdot}5$	-1	20°
Aged 52 . .	$-4{\cdot}5$	$-3{\cdot}5$	
	-0	-1	45°
Aged 56 . .	$-4{\cdot}5$	$-3{\cdot}5$	
	-0	-1	60°

The first is an example of pure swing, and shows how the astigmatism is tending against the rule.

The second shows the change taking place in each eye by a different method. The right eye with astigmatism with the rule has lost all astigmatism and will eventually doubtless show it against the rule. The left eye is achieving the same result by axis swing without any change in amount.

Oscillatory Changes.

In addition to the more or less regular and progressive changes we have noted there is in some patients evidence of an oscillatory change. We examine a patient with the greatest care and order glasses. These are worn perhaps for a few months and then there is a recurrence of discomfort. A slight change is found in the angle of the cylinder which may not amount to more than 10° at most. The new glasses are again worn with comfort and yet again the asthenopia returns. A third examination reveals the curious fact that the angle of the cylinder has returned to its previous position; the original glass is now the correct one.

When we come to the consideration of cylinders $\frac{1}{8}$ D. we may find a change within a month, and it is doubtful whether many eyes are stable within this close limit. It is probable that a small alteration in blood-pressure or in lid tension may from time to time alter the astigmatism within this limit, and that the correct glass depends solely upon the day of the examination. The same applies to small changes in the angle of a weak

cylinder. It is questionable whether we can determine the correct angle of a cylinder under $\frac{1}{2}$ D. within about 5°, and whether most eyes will not vary within this limit value from day to day. Some eyes are more rigid than others and are not affected by slight variations in lid pressure; others are soft and the shape alters with ease. Our experience in cataract extraction teaches us that the hardness of the cornea is very variable. Some cut like butter, others with difficulty.

If these considerations have any value they discount the labours of those who refract to an $\frac{1}{8}$ D. and even less!

We come to the following conclusions:

(1) That the growing eye examined from time to time may show considerable alterations both as regards sphere and cylinder. Astigmatism may alter within wide limits both as regards amount and angle.

(2) That in most cases an equilibrium is attained, and the refraction is stabilised till old age, when a tendency appears towards an increase of hypermetropia.

(3) In other adults progressive changes may be detected, chiefly in the amount and angle of the astigmatism, and that in the majority of cases they tend to replace astigmatism with the rule by astigmatism against the rule.

(4) That in certain cases there appears to be an oscillation of the angle of the cylinder—a pendulum swing within moderate limits.

(5) That few eyes have a permanent refraction within $\frac{1}{8}$ D.

MIDLAND OPHTHALMOLOGICAL SOCIETY, 1921–22.

President :

A. B. Cridland.

Treasurer :

E. H. Harries-Jones.

Secretaries :

E. B. Alabaster.

B. H. StClair Roberts.

REPORTS.

During session 1921–1922 five meetings were held—three in Birmingham, one in Wolverhampton, and one in Shrewsbury. One of the Birmingham meetings was a combined one with the South-Western Ophthalmological Society.

Meeting held in Birmingham, October 4th, 1921.

1. *A case of cyst of the retina.*

By T. Harrison Butler.

There was a single large isolated cyst down and forward.

The President thought that a retinal cyst was somewhat similar to a cyst of the iris, and he regarded it as a clinical entity. He had tapped two ; the fluid which came out was clear, straw-coloured and very albuminous. They had filled up again, but notwithstanding he suggested tapping in this case.

2. *A case of spring catarrh.*

By T. Harrison Butler.

The exhibitor said that such cases were typically seen in the East. They were most successfully treated with radium. In this case the blood-count showed 6 per cent. of eosinophiles instead of 1 per cent.

The President remarked on the crescentic opacity in the upper part of the cornea which was nearly always present in these cases. He advocated the removal of enlarged tonsils as an important part of the treatment.

3. *A specimen of flat sarcoma of choroid.*

By Jameson Evans.

Clinical history.—Woman, æt. 31 years.

R.E.: Detached retina in the lower outer half. May, 1917, diagnosis ? tuberculous choroiditis. Vision, fingers at 3 metres. January, 1921, complete detachment of the retina; neuralgia on the right side of the head. Tension normal. No perception of light. September, 1921, sub-acute glaucoma, severe pain, eye excised.

Section of growth.—Gross: Flat, densely pigmented growth, about 1 mm. in thickness, extending from the region of ora serrata to 4·5 mm. beyond optic disc on the outer and lower side and involving about half the area of the globe.

Microscopically: Corneo-iridic angle blocked. The tumour consists of spindle-shaped and epithelioid polygonal cells of varying sizes and degree of pigmentation. In the central part of the growth the cells are more epithelioid and are arranged in irregular alveolar spaces bounded by fibrous septa. The peripheral portions, and especially the deposit in the perichoroidal space infiltrating the sclera, ciliary vessels and nerves, are more spindle-shaped and show no alveolar arrangement.

Meeting held in Birmingham, December 6th, 1921.

4. *A case of aniridia : bilateral.*

By A. B. Cridland.

Girl, æt. 5½ years, with very light hair suggestive of albinism. Clinically there is no evidence of any iris; there is an anterior polar cataract in each eye. With the eyes directed well upwards the suspensory ligament is well seen. The choroids appear thin, the discs are pale and oval in shape from above down. There is myopic astigmatism.

5. *A case of abnormal retinal vein (? choroido-vaginal) associated with paresis of the left external rectus.*

By A. B. Cridland.

R.E.: An unusually large vein, apparently retinal and in the position of the superior temporal vein, possibly a choroido-vaginal or so-called "posterior vortex vein."

L.E.: Paresis of external rectus.

6. *A case of nearly complete atrophy of the choroid.*

By T. Harrison Butler.

7. *A case of new blood-vessels in the vitreous.*

By Beatson Hird.

A boy, æt. 11 years, sent because of defective vision. No disease or ill-health.

R.E.: Flakes of blood in upper part of vitreous. Mass of new blood-vessels extending forward into vitreous some distance above the optic disc. The vessels terminate in some greyish fibrous tissue.

L.E.: Flakes of blood in lower part of vitreous. Mass of new blood-vessels extending into vitreous upwards and outwards and terminating in greyish fibrous tissue. Tuberculin and Wassermann tests negative.

8. *A case of cockscomb excrescence of the conjunctiva.*

By Beatson Hird.

A little girl, æt. 9 years, attended hospital in May, 1920, with phlyctenular keratitis in the left eye. In November, 1921, for the first time there was noticed a mass of granulations on the tarsal conjunctiva of the upper lid. No enlargement of the pre-auricular gland. Von Pirquet test negative. Her temperature is normal and there is no reaction after an injection of old tuberculin.

In the subsequent discussion Mr. Adams suggested spring catarrh, and Mr. Harrison Butler thought it a case of a low form of tuberculosis.

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9. *A case of persistent hyaloid artery.*

By StClair Roberts.

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10. *A case of congenital aniridia in which there was also a zonular cataract.*

By Fulford Eales.

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11. *A specimen of a lachrymal sac.*

By T. Harrison Butler.

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12. *Three specimens of globes containing lead pellets.*

By Beatson Hird.

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13. *Two neuropathic cases simulating miners' nystagmus.*

By T. Harrison Butler.

There are said to be cases of miners' neurosis without actual nystagmus in which a characteristic lid-twitching

is the cardinal feature of the disease. There is no doubt that there is an almost pathognomic type of lid-twitching, associated with stiffness of the neck, twitching of the neck muscles, and even of the muscles of the trunk, and that it may be impossible to detect actual nystagmus at the time of examination. This has been recognised, and lid-twitching has been scheduled as an industrial disease – as a form of the miners' neurosis. It is, however, very probable that all these men would show nystagmus if examined immediately on leaving the mine, and the symptoms they relate are those of nystagmus. The fact that a miner can claim compensation for lid-twitching apart from oscillation of the eyes makes it essential that we should learn to recognise the characteristic type of twitching, and that any case of a non-miner showing such twitching should be recorded. Cases of this nature are very rare—in fact, I had not seen one till this year. I have, however, quite recently examined the following two cases, which seem to be important in the light of the Compensation Act:

C. B—, female, æt. 15 years, came to the Coventry Hospital on September 14th, 1921. She was suffering from an acute diplobacillary type of conjunctivitis. The vision of each eye was $\frac{6}{4\cdot5}$.

On examination I was at once struck with the fact that the case strongly simulated miners' nystagmus. There was considerable blepharospasm. On looking up the eyes converged strongly and a well-marked nystagmus set in. This was horizontal and like the miners' oscillation, but not quite so regular. There was lid-twitching similar in all respects to that seen in a miner. Had this girl been a miner I should have not had the slightest hesitation in signing her up as suffering from miners' nystagmus.

The treatment was a $\frac{1}{4}$ per cent. solution of sulphate of zinc to be used three times a day in an eye-bath.

In September the symptoms were slight, and on November 16th I wrote for the girl and examined her in

my private rooms. I found that her eyes were normal in every respect.

The strong convergence noted on looking up is not infrequently seen in cases of miners' nystagmus and has been described by several authors.

G. L—, male, æt. 27 years, was sent to me by an insurance company for an examination and report regarding an accident.

On December 4th, 1920, he was chipping a casting when a fragment of metal struck his left eye. He was an in-patient in an eye hospital for three weeks. He started work again on May 2nd, 1921.

On August 18th, 1921, he was grinding castings on an emery wheel and on the next day his eye was inflamed. He surmises that something must have flown from the wheel into the eye, but he is not certain of this.

On September 5th a bar of steel fell upon his foot and he was again laid up for a considerable time.

Examination revealed a condition similar to that found in C. B—. On looking up the eyes converged and the lids began to twitch in a manner strictly comparable to the twitching in miners' nystagmus. Slight irregular nystagmus was noted when the disc was examined with the ophthalmoscope with the eyes turned up strongly. Here again had the man been a miner I should have felt perfectly justified in regarding him as suitable for compensation, as a sufferer from industrial lid-twitching and nystagmus. In addition, there was some spasm of accommodation and amblyopia of the left eye. A retinoscopy without a mydriatic gave variable figures, but all on the minus side, and he refused to take any convex glass. I felt certain that there was some spasm, and under homatropine I found two dioptres of hypermetropia. The vision of the right eye was $\frac{6}{6}$, that of the left $\frac{6}{60}$. There was a linear scar on the left cornea extending to the edge of the pupil, but Placido's disc showed no distortion, nor was there retinoscopic evidence of corneal distortion. The fundi were normal, and the man passed

all the tests for malingering with one eye. I satisfied myself that the eye was really defective in acuity.

This employee showed general signs of neurasthenia and his neuropathic tendency had not been improved by a succession of accidents. He lives at a distance and so I am not able to give his further history. These two cases show that both lid-twitching and a nystagmus comparable if not identical with the oscillation seen in miners' nystagmus may be present in persons with a neuropathic diathesis. They suggest that it was unwise to include lid-twitching as identical with actual nystagmus for legal purposes; and they support those who, I think rightly, hold that miners' nystagmus is developed only in miners with pronounced neurotic tendencies.

Meeting held in Wolverhampton, February 7th, 1922.

14. *Case of optic atrophy with heterophthalmos secondary to sphenoidal sinusitis.*

By Bernard Cridland.

E. M—, æt. 20 years, presser.

History.—First seen on October 30th, 1920, when she complained that the sight of her right eye had been failing for about two months.

(At about that time she had attended once for some slight injury at her work—possibly a foreign body on the cornea.)

On October 30th the condition found was:

Right eye quiet, wide mydriasis, no reaction to light or accommodation, no difference in the colour of the two eyes, no fundus or media changes.

R.V. $=\frac{6}{18}$; L.V. $=\frac{6}{5}$.

Under eserine the pupil contracted well.

November 16th; The right iris had become grey-green, the left being blue; no fundus or media changes.

R.V. $=\frac{6}{24}$. Wassermann negative.

December 1st: V. = fingers at 2 ft. All retinal vessels appear much narrowed. Field much contracted.

December 27th: Indefinite patches of exudate (?) at edges of disc. Nasal examination negative, skiagram negative.

February 16th, 1921: R. pupil noted to be less dilated and in good light was 5·5 mm. and reacting.

March 30th: R. vitreous opacities present, retinal vessels unusually contracted, disc appears redder than left. Pigment changes at and near macula.

May 11th: Complained of L.V. being faulty. L.V. $=\frac{6}{9}$ partly. Eye slightly injected, and a patch of choroiditis towards nasal side of fundus. Field contracted.

A second nasal examination now revealed presence of sphenoidal sinusitis. Sinus washed out, pus found.

Left eye recovered rapidly and with improvement of field.

December 31st: R.V. = no P.L. Cataract present. L.V. $=\frac{6}{5}$.

Features of case.—(1) Indefinite history of injury suggesting, in conjunction with later condition of right eye, the presence of a foreign body in globe.

(2) The association of mydriasis, heterophthalmos, retino-optic atrophy, all secondary to a sphenoidal sinusitis.

15. *Chronic glaucoma at 20 years of age (familial).*

By Bernard Cridland.

A. M—, æt. 20 years.

History.—For one year has complained that her eyes ache and feel weak; occasionally rainbows have been noted around a light at night.

Her brother, æt. 30 years, has been under Dr. Mactier for two years from chronic glaucoma.

The corneæ appear small; measurement is 10 mm. across, R. and L. The anterior chambers are rather deep and the angles of the anterior chambers appear wide.

No fundus changes but the discs show well-marked

cupping, more of a physiological than glaucomatous character: the veins pulsate.

T. = R. 42; L. 42 (Schiotz) (December 24th), falling to 31 in each eye after one month's treatment by miotics.

V. = R. $\frac{6}{5}$ partly $\bar{c}$ + ·50 ⌿ 10° = $\frac{6}{5}$ partly.

T. = L. $\frac{6}{5}$ partly $\bar{c}$ + ·25 sph. = $\frac{6}{5}$ partly.

The fields showed some general contraction all round except below.

(Both eyes have subsequently been operated on by the Lagrange method of sclerectomy with peripheral iridectomy with good results.)

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16. *A case of detached retina in a woman, æt. 45 years.*

By StClair Roberts.

The fundus showed a localised detachment in the region of the macula; it extended outwards for about one and a half disc diameters. There was some slight optic neuritis with a few striate hæmorrhages around the disc. The mass seemed translucent in parts and a number of fine yellow dots were seen on its surface. There was no pain, no redness nor increased tension, vision less $\frac{6}{60}$. The case was discussed by several members and the general opinion was that excision was advisable. Pathological findings will be published later.

The President opened a discussion on "Difficult Cases of Coal-Miners' Nystagmus," followed by Dr. Lister Llewellyn, Mr. Harrison Butler, Mr. Jameson Evans and Mr. Beatson Hird.

17. *Difficult cases of coal-miners' nystagmus.*

By Bernard Cridland.

In the vast majority of cases of coal-miners' nystagmus there is no difficulty whatsoever in making a diagnosis,

but from time to time we are, all of us, I think, confronted with cases in which it is really difficult to say whether men are or are not suffering from this disease.

The knowledge, moreover, that in any given case there already exists a difference of opinion, and by experienced surgeons, too, does not lessen one's perplexities in making up one's mind.

I feel, then, that no apology need be offered for inviting discussion upon unusual cases which present difficulties from a diagnostic point of view, and I hope that a free exchange of opinions will prove of some assistance in securing unanimity in diagnosis.

I venture to here give you two examples of cases which to my mind fall within this category and I hope that others will bring forward cases which have presented equal difficulties to them.

Case 1.—(Hobson), young man, æt. 21 years, married, with one child, first went underground at 14, where for two years he worked as a waggoner. He then at the age of 16 years 4 months joined the Army but was discharged in three months on account of being under age. Returning to the pit he worked for three years as a loader at the coal-face, then in another pit he worked in the main roads at "jagging" for two years. He has suffered from no illnesses.

His symptoms, which had begun with giddiness seven months previously, were as follows: Frontal and occipital pain with giddiness: "everything was on the move in front of his eyes, his head went round if he saw a light, he felt nervous and could hardly stand still, he suffered from insomnia, and when he lay down he felt a thumping at the back of his head. He had lost both appetite and weight."

He was a short, pallid youth, looking ill, but he did not walk in the manner characteristic of cases of coal-miners' nystagmus, although he groped his way along rather ostentatiously.

Almost the whole time during examination he showed what I can only describe as a voluntary spasm of the lids,

for they were screwed up in an apparently purposeful manner, and when asked to look up the purpose became more manifest whilst the eyes were rolled round in an upward direction, and at no time were nystagmic movements noted. Attempts to open the lids were resisted, so that anything like a detailed ophthalmoscopic examination of the eyes themselves was rendered impossible.

It was striking that on asking him to look up no head resistance was present.

As regards the vision, he admitted seeing some white boards placed at 6 metres but nothing else. During the attempt to see the eyes were rolled up.

Case 2.—The second case is also that of a youth, æt. 19 years and 3 months, who had been married for a year and had a child 6 months old. He went underground at 14 and worked until seven months prior to the date on which I examined him.

At that time he received a blow over the left brow and did no work for four months, presumably on account of the blow. Since then he has not worked because, as he stated, he suffered from giddiness with excessive blinking and headache, although on further questioning he said that he did not work because he could not see. He had suffered from no illnesses of any kind.

The picture he presented was that of a pallid undersized youth who did not hold himself in the characteristic attitude of nystagmus cases. He blinked excessively and in a purposeful manner, a tremor of the lids being noticeable when the eyes were closed. On causing him to look up no head resistance was present, but the eyes were rolled around in a manner similar to that seen in the first case.

The vision was $\frac{6}{6}$ in the right eye and fingers at 1 metre in the left.

Under homatropine and cocaine it was possible to obtain a view of the right fundus and no lesion was detected; in the case of the left eye only a fleeting view could be obtained and sufficient to say that no gross

lesion was present. No sign of nystagmus whatsoever was detected even after fifteen minutes in total darkness.

Other cases of this type could be quoted, but these two will serve as examples of abnormal cases in which the diagnosis is uncertain unless you hold the somewhat sweeping view that all miners who present symptoms and signs of neurasthenia should be classified as suffering from coal-miners' nystagmus.

Reviewing these two cases we see that the subjective symptoms in each would do fairly well for a diagnosis of nystagmus, but the signs are characterised by an absence of those usually met with in the disease.

On the other hand, we are confronted with a blepharospasm which is of the voluntary type together with a very great diminution of sight, both of which are characteristic of a hysterical disorder of vision—a condition which, as we know, may occur in any walk of life and in either sex. Let it not be thought, however, that the voluntary type of blepharospasm is not seen in true cases of miners' nystagmus for we are all familiar with such, but I maintain that in these it does not occur without one or more of the usual signs of nystagmus having pre-existed its occurrence. For example, a severe case of nystagmus presenting the usual signs may be followed over a period of two to three years, and towards the end of that time this voluntary blepharospasm may appear. I consider that the true blepharospasm found in nystagmus is an entirely involuntary one.

Turning again to these two cases it is true that both are miners, but we should not allow that fact to have undue weight, and we must at the same time remember that both are very young and have undertaken domestic responsibilities that are better postponed to maturer years.

Had these men not been miners we might have "thought" of nystagmus, but we certainly should not have diagnosed it.

It seems to me that these two cases are examples of a hysterical disorder of vision, or, as Dr. Llewellyn rightly

terms it, a psycho-neurosis having occurred in coal miners.

If we are not to regard them so where else may we look for justification.

In Vol. I of the *British Journal of Ophthalmology* an interesting case was published by Leslie Paton under the title of "Functional Spasm of Accommodation." A soldier who was slightly concussed suffered afterwards from marked spasm of convergence and accommodation, blepharospasm and great limitation of the visual fields; in due course these signs passed away, and the vision (which presumably had been much diminished) returned to normal.

This association of spasm of accommodation with field contraction is of considerable interest, for do we not frequently find it in cases of coal-miners' nystagmus.

Again we may recall the two valuable cases described by Mr. Harrison Butler at our last meeting, and which I hope will in due course appear in print.

Finally, I hope that Dr. Lister Llewellyn, whom we all welcome here cordially, will give us his experience as to the frequency with which this psycho-neurosis or hysterical disorder of vision is met with, and especially after injuries.

In conclusion, Gentlemen, may I suggest that as coal-miners' nystagmus in general is a very wide subject, we confine ourselves in this discussion as far as possible to the consideration of the unusual forms and the difficulties which they present.

Dr. Lister Llewellyn:

Cases of miners' nystagmus may be divided into three groups: the atonic, the spastic and the psychic. In the atonic form the chief symptom and physical sign is a rotatory oscillation of the eyeballs, and the diagnosis is, as a rule, easy and straightforward. In the spastic form photophobia and lid-spasm often make the examination of the eyes difficult and the detection of actual oscillation impossible. In these cases a complete history should be taken and much importance attached to the credibility of the patient.

General examination of the eyes should exclude the possibility of spasm arising from other causes, such as foreign bodies, corneal ulcers and blepharitis. The lid-spasm should be involuntary, increased when light is thrown into the eyes and still present when the attention of the patient is withdrawn by general conversation. I attach great importance to the "head-test." The object of this test is to dissociate the movement of the head and eyes by flexing the head strongly and asking the patient to look up. A sense of head resistance and tremor is felt by the observer's hand placed on the patient's head. In the absence of senile tremor this test is very valuable. In the psychic form, men, after a period of six or twelve months' play, gradually lose the objective signs of the disease, but retain the subjective symptoms, and develop, in addition, a neurosis or psychosis in which general nervousness, mental depression, sleeplessness with disturbing dreams, palpitation, sweating and generalised tremors are present. Actual phobias and abnormal anxiety may also be present. These symptoms, as a rule, develop in the course of the disease and are not marked when the man first gives up work.

The first group is simulated by the various nervous diseases, such as disseminated sclerosis or cerebellar tumour. Miners' nystagmus can be distinguished by the rotatory character of the oscillations, which is very different from the lateral nystagmus, with the quick jerk and slow return, found in these cases. The nystagmus of albinism and of the blindness of infancy resemble the condition found in miners' nystagmus very closely.

The spastic group is simulated by men who have some ocular trouble, such as error of refraction or chronic blepharitis. In high myopia there is often a pseudo-spasm of the orbicularis associated with irregular ocular movements. I have frequently seen nystagmus in high myopes who have never been down a pit. Miners whose sight is failing from any cause naturally attribute their trouble to the onset of miners' nystagmus.

The third group is simulated by men who unwittingly develop a psycho-neurosis to escape from an environment which is distasteful and to which they are not equal.

What is more natural, in the light of our war experience, than that these soldiers of labour in an admittedly dangerous calling should develop a neurosis which shows itself in disturbances of the ocular motor apparatus, especially when this condition not only ensures freedom from the distasteful occupation, but carries with it a solatium in the shape of compensation? These men do not show oscillation of the eyes. Their chief symptoms are loss of sight, headache, giddiness and photophobia. They show marked blinking and possibly generalised tremors. The blinking does not conform with the description I have given of the true lid-spasm. I have seen several cases simulating this group and have successfully appealed in two cases before your President, acting in his official position of medical referee for this district. The men are young, often recently married, the mental symptoms are present at the period of failure and they are rarely coal-face workers.

But even when you have made a correct diagnosis your difficulties are not at an end. Men getting on in years or men who from the onset of some chronic illness are unable to continue their work, attribute, with perfect good faith, all their symptoms to the onset of nystagmus, which becomes the third partner of the disease.

When the old age pension scheme first came into operation in Ireland the number of men and women who, with the cheery optimism of their race, thought they had reached the promised land, created an embarrassment in the local exchequers and upset the calculations of all the actuaries. So at the present time the number of men who, feeling their working days are approaching an end, make their financial position secure by obtaining the certificate which qualifies for compensation, is large and increasing. Let me give you two examples:

J. B—, æt. 64 years, while proceeding to work collapsed and was brought home. During the next few days he nearly

died from an acute attack of bronchitis complicated by mitral disease. He sent in a claim to the company for "bronchitis brought on by work in the pit." The claim was ignored. His doctor then found he had nystagmus and the man was eventually certified. I appealed against this certificate on the ground that, although nystagmus was present, the incapacity was due to his general condition. The medical referee held that he had only to decide if nystagmus were present or not.

W. T—, æt. 70 years. A case of paralysis agitans brought a certificate to me last week.

The average age of my last fifty cases is over 52 years, the normal average being 42.

It has been shown that at least 20 per cent. of all men who have worked in a coal-pit for ten years or more show nystagmus on examination. This nystagmus in the great majority of cases causes no inconvenience and entails no loss of wage-earning capacity. At a certain North Staffordshire colliery it was found necessary to shut down the afternoon shift and 250 men were discharged. In the period July to November, 1921, 23 of these discharged men obtained a certificate for nystagmus. Every one of these men had applied more than once for work. No fresh cases developed from the 400 men who continued working during this period.*

None of these men, with one possible exception, were malingerers, and all would have continued to work if allowed to do so. True malingering is very rare, but conscious exaggeration of symptoms is almost universal. The economic problem involved is a very serious one and the wastage of man-power is unnecessarily great. It should not be assumed that because a man shows the objective signs of nystagmus he is necessarily incapacitated, and for ever after incapable of working underground.

Conclusion.

The present schedule definition of the disease is a broad

* *First Report of Miners' Nystagmus Committee*, p. 24.

one, and opens the way for the inclusion of cases which are not those of miners' nystagmus. The certifying surgeon should not make the diagnosis of the disease on the history given by the patient unless there is confirmation by physical examination. Rotatory oscillation of the eyes; true lid-spasm associated with photophobia; head tremor with backward inclination and resistance of the head, combined with objective giddiness; may separately be considered sufficient to confirm the diagnosis when accompanied by a typical history. Irregular movements of the eyes, rolling of the eyes about, blinking, may all be purposive. Above all it is important to realise that a miner may develop a psychoneurosis quite apart from his underground life, and although he will probably refer his symptoms to his oculo-motor apparatus, it by no means follows that the pit work is the cause of his incapacity. In all these cases the certifying surgeon must satisfy himself that objective signs are present and not grant a certificate on the man's history alone. If he does he will confirm the man in his neurosis and do him an unintentional injury.

Mr. Jameson Evans

thanked the President and Dr. Llewellyn for their kind exposition of a very difficult subject. It seemed to him that the problem of nystagmus was becoming increasingly difficult for the surgeons and assuming alarming proportions from the economic point of view.

Time was when the decision as to whether a patient did or did not suffer from nystagmus appeared to be a fairly simple matter, but now, with tremors and spasms, photophobia and possibly other neurotic symptoms forming part of the symptom-complex, it was often a very difficult and tiresome matter to formulate such a definite diagnosis that it could be upheld and substantiated in court.

The elimination of involuntary oscillations of the eyeballs as an essential feature of nystagmus had robbed

us of the most valuable diagnostic sign of genuine nystagmus, and we were often compelled to form an opinion on our personal view as to whether the accessory signs and symptoms were genuine or not. Head tremors and spasm of the neck muscles would appear to be difficult to simulate, but convergence, lid-spasm and photophobia were easily imitated, and the term "characteristic" could hardly be applied to them. All these signs might well be perfectly imitated, consciously or otherwise, by men who have every facility for observing and studying the genuine disease in their fellow workers. He felt not a little suspicious of cases which might be described as purely "spastic."

The introduction of a "psychic" type (without oscillations of the eyeball) still further added to our difficulties both as to diagnosis, ætiology and prognosis. He assumed that psychic nystagmus would only be diagnosed when the psychic symptoms were associated with or immediately followed atonic nystagmus. Unless this were so neurasthenia among miners would have to be regarded as an industrial disease at all times.

He thought it highly desirable that some method should be devised to enable the surgeon to estimate whether oscillations of the eyeballs were or were not a cause of incapacity for work. It was obvious that in a large majority of cases oscillatory nystagmus did not give rise to incapacity or even to any depreciation of wage-earning capacity, yet it was open to any worker who had oscillations of the eyeballs, of which he had been well aware, to claim compensation at any time when it might suit his convenience, and as far as he knew there was nothing to guide the surgeon as to whether the patient's statements as to incapacity for work were true or not. He thought that this fact was well known amongst the miners, and use had been made of it during strikes and periods of industrial slackness in the mines.

He was not greatly impressed with the idea that errors of refraction were a very potent factor in the production

of nystagmus or its rate of recovery. Certainly many emmetropes suffered from the disease and their rate of recovery did not appear to him to be more rapid than that of ametropes, though he could not supply statistics to prove it. He was firmly convinced that recovery was delayed in cases where occupation of any kind was denied to or avoided by the patient. These cases appeared to be just the cases likely to develop psychic symptoms and become chronic invalids. The general health of the patient was an important factor both in the establishing of incapacity and in the promotion of recovery. He hoped that some definite *régime* would be adopted and applied in every case of nystagmus so that the patient's mind should be diverted from his complaint, and his mental and physical energies directed to other and more salutary channels.

Mr. Harrison Butler

said that he would never diagnose miners' nystagmus unless there was definite nystagmus of the typical kind. This was not always easy to elicit. He made the miner look at his eyebrows for a long time and while he was doing it he held both hands behind his neck. The neck resistance and tremor were typical of the disease and he agreed with Dr. Llewellyn that it was a valuable sign. If these procedures did not cause nystagmus he left the miner in a coal cellar in the dark for half an hour and then examined him with the ophthalmoscope, with his head bent forward so that the eyes turned up. Slight nystagmus was often seen when magnified in the ophthalmoscopic picture. He felt certain that in most of the cases in which the miner gave a typical history of the symptoms, even when it was impossible to elicit nystagmus, it would be found if the man were examined immediately after leaving the pit, but such a case could not be signed up. Whereas the typical lid twitching had been officially scheduled, he was obliged to sign it up even when he could not see any actual nystagmus, but he felt

quite certain that these men had real oscillation of the eyes in the pit. If a man had had a dose of alcohol before the examination it in some cases abolished the nystagmus. He had not had any experience of malingering. He found that it was very difficult to get miners to give up the colliery for other work, and he was certain that malingering was very rare under normal conditions of working.

Mr. Beatson Hird

expressed the opinion that the neurotic symptoms were a super-added condition. He asked that there should be agreement as to what signs should be present for certifying the presence of the disease, so that there should be unanimity as to fact and thereby avoid disagreement in certification and reports.

An invitation having been sent to the South-Western Ophthalmological Society, a joint meeting was held in Birmingham on April 4th, 1922. The proceedings commenced in the morning with operations performed by members of the staff of the Birmingham and Midland Eye Hospital.

Mr. Jameson Evans operated on a case of chronic glaucoma, doing a Herbert wedge isolation.

Mr. Harrison Butler, using a point of light illumination, needled for capsule. He also excised a lachrymal sac under local anæsthesia.

In the out-patient department a lantern demonstration of "Sweet's eye localiser" was given.

18. *A case of hole at the macula due to a war injury.*

By Jameson Evans.

There had been a rapidly-moving missile in close proximity to the globe.

19. *Two slides of glaucoma.*

By Jameson Evans.

20. *Slide and photograph of case of epithelioma in the filtration scar of a Lagrange operation.*

By the President (A. B. Cridland).

21. *Two cases of plastic operations by the Esser method.*

By T. Harrison Butler.

In one of them, a case of very severe burn with extensive scarring of the skin of the forehead and cheek, it was quite impossible to think of turning down flaps, because the skin was like parchment. Eight operations had been performed, which included two Esser operations. The final result was very gratifying, and the girl could now wear an artificial eye and take her place in society.

The second case was brought forward as an illustration of the fact that occasionally the Esser pit lined with the graft does not completely unfold.

The condition of severe ectropion caused by extensive cicatrisation after a cellulitis had been completely relieved by a pedunculated flap followed by an Esser out-lay. The latter had left a small slit-like pit lined with skin. It was proposed later on to perform a small operation to allow this to unfold.

22. *Case of limbal tumour.*

By T. Harrison Butler.

He had hoped to show a similar case which had been completely cured by radium irradiation. It was considered to be a limbal sarcoma. The present case had the following history :

A month before coming to the Birmingham Eye Hospital it was noted that a white swelling had appeared upon the left cornea. This gradually grew to its present size.

The patient, a girl, æt. 19 years, shows on the outer aspect of the cornea, and somewhat invading the sclera, a flat, yellowish-white, slightly-raised patch. It is about 6 mm. in diameter, and is very slightly injected at its outer aspect.

Mr. Jameson Evans and Mr. Harrison Butler considered that it was perhaps a malignant tumour, and suggested that it be treated by radium at the Radium Institute. The authorities there had some doubt as to the malignancy of the patch, and invited Mr. Holmes Spicer to give an opinion upon the case. He considered that it was a dermoid cyst.

Against this theory is the fact that all the girl's relations, and she herself, are quite confident that the growth is of recent appearance.

The case was discussed by the meeting, and three diagnoses were considered: it might be malignant; it might be inflammatory, perhaps tuberculous; and finally it could be a dermoid.

The general opinion was that it was not malignant. Some members inclined to the inflammatory theory, but the majority held that it was a degenerating dermoid.

Since the date of the meeting the growth has gradually become more injected and decidedly more yellow. It has had one irradiation with radium at the institute. The present effect of this treatment has been to stimulate the condition. It now looks decidedly angry, and there is considerable general irritability of the eye with photophobia. The growth is now quite yellow, and it is growing both in height and extent. The nature of the disease is still *sub judice*.

23. *A case of keratitis.*

By Beatson Hird.

A female, æt. 41 years, attended the Birmingham Eye Hospital on January 5th, 1922, and complained that her eyes began to trouble her just before Christmas last, there

being a feeling of something in her eyes, and the sight became misty.

She presented the same appearance as now, but not quite so marked.

There were no inflammatory signs present, and her vision was—right eye $\frac{6}{18}$ and left eye $\frac{6}{36}$.

The cornea did not stain with fluorescein either then or on any subsequent occasion.

Each cornea in the central area shows a number of short, separate, rather broad, sinuous opacities in places, causing elevation of the surface.

The condition has increased, and vision is now—R.E. $\frac{6}{24}$ one letter, and L.E. less than $\frac{6}{60}$.

Mr. Richardson Cross said it was an interesting and unique case. He thought there was some low form of infection which had gained entrance into the *substantia propria*. He suggested subconjunctival injections.

Mr. Roper suggested ionic medication with zinc or mercury.

Mr. Hay had had a similar case which did not yield to treatment with the usual remedies.

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24. *Discussion on heterophoria.*

Mr. Richardson Cross:

In the year 1887 I attended the International Congress at Washington, when a paper was read by Dr. George Stevens, of New York, on "Some Important Problems Respecting Insufficiency of Ocular Muscles."

Advances in the knowledge of the anomalies of the directing ocular muscles had not kept pace with those made on errors in refraction and accommodation. Graefe had taught the doctrine of "muscular asthenopia," which he attributed to insufficient force of the interni to overcome the resistance of the externi towards the reading point. He had introduced the dot and line test, and had shown this weakness to be present in many cases.

At that time the study of muscular irregularities (excluding strabismus and the results of paralysis) had been almost exclusively that of "insufficiency of the interni" at the reading point—although other insufficiencies had been observed. But, as Stevens pointed out, insufficiency of the interni is not the only error of this type; it is not the most important nor is it perhaps the most frequent.

When the eyes are directed straight forward at a point about 6 metres in front of them in the same horizontal plane they are in a state of minimum innervation; this is the condition under which any anomalous tendency of the ocular muscles would be more truly shown.

He alluded to the fact that the same patient might have a "tendency in" for distance, while in reading the visual axes might wander "outwards"; other anomalous results might be encountered in the investigations of these deviating tendencies under varying circumstances. The irregular phenomena would result according to the tension present in a certain muscle or group of muscles.

Slight errors frequently gave rise to nervous irritations and sought for relief. One important element often present in the cases, when symptoms seemed contradictory, was that condition of the ocular muscles in which the direction of the visual axis of one eye showed a tendency to rise above the direction of the axis of the other eye. And to this condition of *hyperphoria* he attributed many of the cases most difficult of explanation.

Ever since this time I have been interested in this subject, which I soon realised to be one of great practical importance, especially in regard to the relief of headache and eyeache which accurate correction of the refraction errors had failed to relieve.

Dr. Stevens kindly sent me his papers from time to time, "Anomalies of the Ocular Muscles," etc., worked out in much scientific detail. I brought back from the Washington Congress and still use the very "phorometer" which he made use of for his demonstrations there.

Meanwhile, others have further elucidated this important subject, and have done much to improve the methods of investigation. Notably, Ernest Maddox by his writings, his work on prisms, and the introduction of the double prism, the Wing test, and especially in the use of the rod or bar.

Observations of the gradual development of the sense of vision in a child from birth show a continuous process in the power of perception, and of the conscious movement of the eye towards the object looked at.

The movements are at first spasmodic, irregular and apparently purposeless. At about a month old one or other eye will tend to fix an object, and will gradually keep fairly steady upon it. Later the two eyes will work in unison.

Very young children often apparently squint; their fixation power may be fairly good, but the co-ordination of the eyes is imperfect, the fusion faculty is not developed. At about six months fusion combines the two eyes in binocular fixation, and later with more accurate muscle balance the eyes follow a moving object together.

At five or six years the power of vision is good and the fusion sense probably does not develop much after this age.

An important control over binocular and perfect vision must lie in the complex but splendid arrangement of the nerve-paths and centres along which the visual impulses pass from the retina to the occipital lobe.

The optic nerve-fibres from the right or left halves of each pair of retinæ are associated with the right or left occipital lobe.

The upper fibres pass along the upper portion of the optic nerve tracts on to the thalamus, and later reach the upper part of the calcarine area of the occipital lobe. The middle fibres pass along to the cuneate and lingual gyri.

Some of the fibres, possibly *the superior ones*, go through the pulvinar to the outer surface of the occipital

lobe and to the angular gyrus. The functions of all are blended together in the effort to obtain perfect sight.

Without tracing the exact paths it may be said that the surface of the retina is represented around the calcarine fissure and in the occipital pole, and that here the field of vision is spread out apparently in absolute reproduction of the retinal areas. The sight here perceived must have a great controlling influence over the proper direction of the eyes in the focussing or regarding of objects in the outer world.

The tendency to the central proper fixation of the eyes is congenital. The active macula is soon made use of by the child at a few months old—he learns to rely on its information, and later learns to refer other objects to their proper position in the visual field. The fusion sense becomes stronger, the habit of binocular vision is acquired and elaborated, and in return the co-ordination of the nervous impulses and of the muscle balance increase in efficiency.

When the muscles are strong and normally innervated, and the fusion sense is well maintained, with a strong desire to co-operate between the two occipital regions in which the retinal pictures of the two visual fields are represented, binocular vision is maintained even under difficulties.

Even in some cases without any help from the control of fusion, the anatomical balance by mere natural tension of the muscles keeps the two visual axes concomitant and normal in position.

The direction of the visual axes is usually correct. If faulty it is recognised as *squint*, or realised by the patient as *diplopia*.

The normal direction of the axis of the two eyes is maintained by a proper balance of the orbital muscles controlled from the nuclei in the mid-brain. But they also come under the influence of higher brain centres, nerve impulses passing through the optic radiations by the brachium superius into the ocular nuclei.

The static balance of the eyes depends on the muscles, their origin, insertion, length and elasticity. The muscle fibres are subject to the nerves supplying them, and to the varying nervous impulses which pass through the nuclei reflexly or from more distant portions of the optic tracts.

The eyes are moved into and maintained in the positions needed by certain of the muscles, which are steadied and resisted by the elasticity of their opponents.

The position of the eyes after death or under chloroform is usually in some degree of divergence, but when active, though at rest, usually show a tendency to slight tonic convergence.

Either the diverging or the converging muscles may be a little the stronger in any given case, and this preponderance gives a tendency to the position of the eyeballs; but usually the proper position for binocular vision is easily maintained.

In normal binocular vision the visual axis of each eye passes from the object looked at to the fovea—the images on the two maculæ become perfectly blended, and the properly associated areas of the two retinæ are superimposed.

The perfect position of the two eyes is maintained in proper balance by the ocular muscles. In most cases there is a perfect equilibrium of motor balance together with normal nerve control under the varying directions and movements of seeing. But the balance is not infrequently faulty more or less from muscular defect.

In slight insufficiencies binocular vision may be usually well maintained and without discomfort; but under conditions of weakness or ill-health the muscular insufficiency gives rise to strain, and suitable tests may show the faulty tendency. In other cases binocular vision is difficult to maintain; a greater or less reflex effort is needed in order to keep up the proper position of the visual axes.

The desire for binocular vision associated with a strong fusion sense stimulates an unconscious effort to maintain

proper position of the eyes, but may cause more or less discomfort.

A strong fusion sense will help to overcome a definite faulty tendency or inequality of muscular balance, but at the cost of some eye-strain or muscular asthenopia; while at times, or in other cases, the faulty tendency is too great to be overcome by the balancing power, and a temporary squint may be the result, this being the more probable where there is a weakness of fusion power. Even if such squint is not manifest its tendency can be shown to be present, for when one eye is screened from the object looked at by its fellow, and watched, it will be seen to wander whither the muscular balance takes it.

The position of static equilibrium (the eyes fixed at distance, the position of the visual axes practically in parallelism) is the starting-point in testing for heterophoria.

When the position of the eyes and the influences that maintain binocular vision are normal the condition is called "orthophoria," but when there is any tendency to the positions of the visual axes being out of line, or either eye tends in the least to wander, any such faulty tendencies are grouped under the term "heterophoria."

The usual primary positions of the eyes are less liable to instability than are the secondary ones: their balance is automatic, strengthened by habit, and governed reflexly from the lower basal centres, while unusual secondary positions are guided by volition and controlled from the higher nerve centres in the brain.

Although under good conditions the eyes are straight and see normally, enervating causes and ill-health weaken nervous control, and if there is a slight tendency to faulty position of the eyes or faulty balance of the muscles, such defect may now become manifest by disturbance of vision or discomfort. We know that occasional squints or double vision can develop during fevers or other forms of ill-health—during the teething of children, etc.—and in some cases squints recur periodically.

Now although typical squint is essentially a defect due

to weak fusion power, and heterophoria is essentially dependent on muscular insufficiency, the two conditions in my opinion overlap one another. They pass from one into the other : the very existence of heterophoria is shown and proved by tests which demonstrate a condition of diplopia or want of symmetrical fixation. And how are we to designate the conditions which lie between a manifest diplopia or squint and a latent tendency to it, which is indeed heterophoria.

Some patients show curious anomalies in the pose of the head, or in the expression of the facial muscles ; and such may be associated with deficiencies of the ocular muscles or with errors in the declinations of the eyes, or of their tendencies up or down. These may be accounted for by the close relationship of the anterior longitudinal bundles, and of the lemniscus or fillet with the corpora quadrigemina and the ocular nuclei in the mid-brain.

Mr. Jameson Evans :

I am glad to find that Mr. Richardson Cross has fully discussed the wider and complex subject of binocular vision, and I shall therefore restrict my remarks to heterophoria in its limited sense of latent deviations which are comitant in character. In this way I shall avoid the discussion of noncomitant heterophoria due to paresis or aberrant insertion of muscles, where binocular vision is maintained in the ordinary way, but the amount of latent deviation varies in degree in different parts of the field of fixation. Further, I have no remarks to make on cyclophoria.

It appears to me that most of the cases of heterophoria we have to deal with are lateral deviations—eso- or exophoria. Hyperphoria must be regarded as more or less of a separate entity, but its features certainly seem to be modified to a great extent by the state of balance of the convergent and divergent functions. As convergence plays such an important part in the etiology and

symptomatology of heterophoria I should like to refer to the physiology of convergence.

Following Maddox's description we recognise three elements of convergence:

(i) *Tonic convergence*, by means of which the visual axes are brought to practical parallelism, so that on viewing a distant object, and occluding one eye, it either remains undeviated or only deviates slightly.

(ii) *Accommodative convergence*, which is due chiefly to sympathy with accommodation, but partly also to the habit of converging when attention is directed to a near object. Its amount depends upon the amount of accommodation required. As a rule each dioptre of accommodation is accompanied by about three-fourths of a metre angle of associated convergence (*e. g.* 4 D. accommodation with 3 m.a. convergence).

The deficit of 1 m.a. is made up by the third factor, viz.:

(iii) *Reflex convergence* or fusion convergence, by means of which the two images brought together by the first and second factors are fused into one. Its reality is shown by the fact that in fixing a near object there is a (physiological) deviation of the eyes if the desire for fusion is suppressed by screening one eye or by rendering the images dissimilar or widely separated. This visual reflex which co-ordinates (i) and (ii) is a very important factor in heterophoria, since it is constantly in action during waking hours, and being a later development of higher evolution is liable to give rise to symptoms of ocular fatigue which can be relieved by the wearing of abducting prisms.

A disturbance of any or all of these three convergent factors may give rise to distressing symptoms which are generally grouped under the vague term of "muscular asthenopia." On the other hand heterophoria may not cause any symptoms, and then there is no occasion to deal with it therapeutically. Where symptoms of muscular asthenopia appear it is necessary to inquire which of the

three factors is chiefly at fault. For this purpose we test the amount of accommodative power by such means as the hair optometer or small reading type, the power of adduction and abduction by means of the prism-verger, the latent convergence or divergence by means of the Maddox rod for distance, and his wing test for near (in both cases with the refractive correction worn).

It is generally regarded that the normal convergence near-point should be 5·5 cm. except in presbyopia, where it may be 8–9 cm., and anything more remote than this is indicative of convergence insufficiency or divergent strabismus. In the main, however, we are guided as to diagnosis and treatment by the results of the "rod" and "wing" tests and the following rules:

"A deviation which increases notably in near vision indicates in general an anomaly of convergence, and a deviation which is essentially greater for distance than for near points to a divergence anomaly."

Whether these tests as applied during one examination give us the true amount or kind of latent deviation I am not in a position to state, but it has been suggested that we might get very different results after prolonged atropinisation or after exclusion of one eye for several days.

It is generally considered that an esophoria may be due to convergence excess or divergence insufficiency, but if both conditions co-exist we get an esotropia—at any rate after a time. The same remark applies to exophoria due to divergence excess or convergence insufficiency where the two conditions combined would produce a divergent strabismus.

According to Sherrington's law, excess of stimulation of one movement produces inhibition for the centre of the opposing movement and *vice versâ*, and from this we might argue that convergence or divergence excess would inevitably lead to weakness of the opposing action and therefore to the combined actions resulting in the development of squint, but I do not think that this happens in most cases of heterophoria in adults. As to the causes of these

anomalies of convergence and divergence, we appear to be agreed and definite as to some cases and very indefinite and doubtful as to others.

(a) *Convergence excess* is usually accommodative, *i. e.* due to forced accommodation as in hypermetropia, astigmatism, feeble accommodative power, medial opacities, bad illumination, and in wearing minus glasses for the first time.

A non-accommodative form is described and ascribed to hysteria and "conditions producing spasm"—both very vague expressions. Perhaps these are due to reflex irritation through the cerebrospinal or sympathetic systems as exemplified by teething and intestinal worms in children.

(b) *Convergence insufficiency* is also often accommodative, *e. g.* in myopia. We occasionally find it in hypermetropia and presbyopia when using plus lenses for the first time. Convergence may be relaxed in those who cannot see well for near, *e. g.* in presbyopes, higher hypermetropes and anisometropes, and in such cases convergence insufficiency may be relieved by the use of plus lenses.

A non-accommodative form of convergence insufficiency appears to have a genuine existence, and may be due to traumatic neuroses, hysteria, neurasthenia, myasthenia, debilitated conditions and septic infections—particularly those due to nasal sinusitis. If we regard primary divergence excess as a reality, convergence insufficiency would probably be associated with it.

Divergence excess as a primary condition seems hard to realise apart from a condition where the externi have a mechanical advantage, *e. g.* in wide orbits, and I should say that it is generally secondary to convergence insufficiency.

Divergence insufficiency is also usually secondary to convergence excess, except where the externi are acting under a mechanical disadvantage. As to the symptoms of heterophoria, there is nothing definite to distinguish those of muscular asthenopia from accommodative asthen-

opia. Pain in the eyes and head, conjunctival irritation, blurring of vision after close work are common to both, but occasional diplopia, giddiness and dizziness, digestive and general nervous disturbances, especially when they arise after observing objects in motion, may be regarded as symptoms of failure to maintain binocular vision and exhaustion of the convergent and fusion centres. This exhaustion seems pronounced in cases where the difficulty of fusion is in the vertical meridian, and for this reason the latter symptoms are often found with hyperphoria.

Treatment of heterophoria resolves itself in the main into the correction of convergence excess and insufficiency and their causes. To a great extent this applies even to hyperphoria. The methods adopted include—correction of error of refraction. Constitutional treatment, including the eradication of septic foci. General tonic treatment undoubtedly influences the three convergent factors and particularly the amplitude of the fusion-convergence reflex. Prisms may be used to exercise the muscle or muscles which appear to be underacting. Exercises without prisms, *e. g.* convergent exercises, by approximating some object towards the eyes whilst maintaining binocular vision, or divergent exercises with the amblyoscope, may be employed. The use of prisms for the relief of an underacting muscle must be resorted to when exercises are not applicable or have failed in their effect. Under these circumstances we must recognise that they are of the nature of a crutch, and a tacit admission of failure, on our part, to cure the defect. Still the *defect per se* is of no consequence as long as it does not give rise to any ill-effects. It is pretty well recognised, I think, that the majority of latent deviations call for no treatment at all. Hyperphoria causing distressing symptoms can often be treated successfully by means of relieving prisms, but even here the correction of refractive errors is of primary importance. Prism exercises are seldom tried. I have no experience of operative measures for the relief of symptoms of heterophoria. I cannot recall a case of

comitant heterophoria in which such a procedure seemed called for.

Mr. E. H. Stack (Bristol)

said he would confine his remarks to hyperphoria. He had been through his last 2000 cases and was surprised to find 80 in which there was one degree or over of this condition, and in 42 of these he had given prisms—that was, in about 2 per cent. of all his cases. There were five women to two men—75 per cent.—between the ages of 25 and 50. He had written to those whom he had not seen since prisms had been ordered, or not ordered, for a candid opinion as to whether they were comfortable or not. About 10 per cent. of those who had not been given prisms complained of some discomfort, while those who had had prisms practically all said they were satisfied. It was difficult to lay down rules for ordering prisms. In those who had not been wearing glasses, unless over $1\frac{1}{2}$ degrees, he did not order prisms the first visit. With one eye to some extent amblyopic he depended chiefly on two points—that diplopia could be fairly easily induced by prisms or coloured glasses, and that the correcting prism gave a very definite sense of comfort both for distance and reading.

He never ordered prisms in those cases in which there was orthophoria when under atropine, but he would like to know the practice of others on this point. When the patient was much run down it was better to procrastinate.

As to causation, it was an acquired condition. It was certainly uncommon in children. Sixteen was his youngest case, though he admitted he had not examined in the school clinics as a routine. It was also uncommon without a fairly definite amount of error in refraction, and nearly always with more than a trace of astigmatism; many had a definite debility of some sort and the symptoms and necessity for prisms in these cases often disappeared, though the hyperphoria generally remained and was pretty constant in its amount.

He thought the imbalance was a reflex from local con-

ditions in the eye often aggravated by various other factors. To mention a few, he had found it in two cases of diabetes, in one of Graves's disease, in two of early cerebral tumour, and in at least one of sinusitis. He had not found it as a cause of migraine. In one case the pain had been so severe that the patient had been treated for some time for supra-orbital neuralgia.

Mr. Harrison Butler

said that using the Maddox wing-test he was finding that a large number of presbyopes also showed considerable convergence insufficiency, and were made very comfortable by prisms base in up to a total of 4°.

He emphasised the fact that esophoria and exophoria were liable to great variations. It was quite common to treat a large heterophoria with prisms, and to find at a subsequent examination that the defect had disappeared and that the prisms were now causing discomfort. For this reason these cases of heterophoria should be examined frequently. This did not appear to apply to hypophoria in which the deviation found with the Maddox rod was apt to be permanent. These patients with hypophoria were grateful for prisms up and down.

Mr. Hay

commented upon the fact that it was considered necessary to test for heterophoria with corrected vision if really scientific work was to be done. With the present spectacle frames, however, it was not possible to centre the trial lenses in a vertical direction for both eyes simultaneously, and whereas on the one hand the amount of hypophoria was usually a small one, on the other hand, the vertical separation between the two pupils might be a matter of several millimetres, and the decentration of one of the lenses by such an amount, especially in the higher powers, would obviously lead to error in estimating the amount of hypophoria.

The provision of better trial frames for these tests was becoming of increasing importance.

Mr. Beatson Hird

said that he had found there were double the number of females presenting heterophoria as males. On looking through his notes he found 17 per cent. of exophoria of over 2 prism dioptres and he had ordered prisms in 3 per cent. of these cases. If the convergence was good he did not order prisms.

He had found only 5 per cent. of hypophoria of over 2 prism dioptres.

A large percentage of heterophorias falls between the ages of 41 and 50. He found that hyperphoria was often much less for near than the distance.

The President (Mr. Cridland),

in summing up the discussion, said that optometry was as much an art as a science and one must estimate the personal factor in each case. He was always cautious in the prescribing of prisms, and considered an accurate correction of the refraction a most important point.

Meeting held in Shrewsbury, June 13th, 1922.

25. *A case of traumatic iridodialysis.*

By Russ Wood and F. A. Anderson.

26. *A case of tuberculosis of the conjunctiva.*

By Russ Wood and F. A. Anderson.

Mr. Russ Wood said he had tried calcium collosal, also he had given T. O. every eighth day in doses from ·0001 to ·01 mgrm. also guaiacol subconjunctivally.

The President said he gave T.R. in doses of $\frac{1}{10000}$ mgrm. every second day and continued the treatment for two months.

Mr. T. Harrison Butler suggested diathermy or radium.

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27. *A case of posterior lenticonus with connective-tissue changes at the disc.*

By Russ Wood and F. A. Anderson.

28. *Two cases of epithelial inlays for contracted sockets.*

By Russ Wood and F. A. Anderson.

The results were good, the patients being able to wear their artificial eyes quite well. The skin had been taken from the inner surface of the arm.

29. *A case of choroiditis in both eyes at the macula simulating familial choroiditis.*

By Russ Wood and F. A. Anderson.

The case was discussed by the President (Mr. A. B. Cridland) and Mr. T. Harrison Butler, who thought it might be congenital, but Mr. Russ Wood pointed out that there had been considerable improvement under treatment with mercurial inunctions, diaphoresis and a mixture of mercury and potassium iodide.

30. *A note on two cases of cervical ribs with eye symptoms.*

By T. Harrison Butler.

E. M. T—, æt. 20 years, female, came to the Warneford Hospital in December, 1921. She complained that two years previously her pupils had become unequal, and that from this period she had suffered from headache.

Examination showed that, apart from the pupils, both eyes were perfectly normal with no refractive error. The right pupil in normal daylight measured 3·5 mm., the left 6·5 mm. Both pupils reacted normally. Cocaine instillation had no action whatever upon the right eye, whereas the left dilated *ad maximum*. Horner's syndrome was present. In addition to the contraction of the pupil there was slight ptosis and some enophthalmos. The right side of the forehead was dry, the left moist. The girl flushes more on the left side than on the right.

A careful examination was made to trace a cause for this complete paralysis of the right cervical sympathetic nerve. There was a saddle-nose, but this was due to falling upon

the fender in childhood. The Wassermann was negative. I could find no history of tuberculosis, nor did the physical examination afford any evidence of phthisis.

The idea flashed through my mind that this might be a case of cervical ribs, and the X-ray picture confirmed this guess.

The skiagram shows a typical cervical rib on each side. As there was no neuralgia or other symptom of an urgent type, it seemed to me that it would be quite unjustifiable to perform any operation for the removal of these extra ribs, the more so because no improvement could be expected in the sympathetic paralysis.

The second case had quite different symptoms. It is curious that she came to see me privately two days after I had examined E. M. T—, and it was this coincidence which led me to the correct diagnosis.

Miss M. W—, æt. 33 years, complains that she has a peculiar feeling in her head. Her nerves are wrong. Her eyes ache, the right more than the left. Her right eye cannot stand the light, especially early in the morning. She has severe neuralgic pains in the right side of the neck, and these radiate down the right arm. All these troubles followed influenza three years ago.

There was no error of refraction or muscle balance, and both fundi were perfectly normal. There was no history or symptoms of any nasal trouble, and the blind spots tested on Bjerrum's screen were normal in size. The fields of vision were not diminished. Physical examination of the nose showed no obvious abnormality. The right eye is slightly injected and flushes easily.

In the light of the last case, I thought that there might be a cervical rib pressing upon the brachial plexus and irritating the cervical sympathetic. An X-ray picture showed that this was the case.

Miss M. W— went to Edinburgh and I think consulted Mr. Sym. This is a case in which, because of the pain, it would be quite justifiable to remove the ribs. I believe that the operation is a formidable one.

NORTH OF ENGLAND OPHTHALMOLOGICAL SOCIETY.

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REPORTS.

Meeting held at Manchester, October 14th, 1921.

1. *Visual fields in lesions of the nervous system.*

By J. Gray Clegg.

Dr. J. Gray Clegg, of Manchester, read a paper on visual fields in lesions of the nervous system. Of 20,000 patients he had made a charted record of the fields of some 900 individuals—that is, about 4·5 per cent. He confined his attention chiefly to those connected with

affections of the nervous system. After showing slides illustrating the visual paths and centres, charts of the defects noted in papillœdema as such, those in primary optic atrophy and retrobulbar neuritis were exhibited. There were several diagrams of the results of trauma of the optic nerve, of which two showed entire loss of an altitudinal half of the field, one a central absolute and another a paracentral absolute scotoma. Various effects of pituitary disease were described. He also produced hemianopic fields; those of right-sided loss were associated with mind-blindness in one and word-blindness in two cases. Quadrant anopsia was shown twice, once of an inferior and once of a superior type. He had also come across one case of hemichromatopsia and word-blindness. Another remarkable field presented a small remaining central area of sight after a left hemianopsia in 1903 and a right hemianopsia in 1911. An interesting case was that of a patient with inferior altitudinal hemianopsia affecting both fields. One illustrated the changes in a case of tumour of the left auditory nerve. Lastly, the peculiarities of fields of vision in the psychoneuroses were exhibited.

Meeting held at Sheffield, November 11th, 1921.

2. *Abnormalities of the ocular muscles, and their treatment.*

By G. H. Pooley.

Mr. President, Ladies and Gentlemen,—I have chosen for my subject to-day the various deviations of the ocular axes usually found in practice.

I will begin with what are commonly called muscle errors as found in the ordinary routine examination.

A few people have no muscle error, but the commonest position for the eyes at rest, as determined by the Maddox rod, is one of slight latent convergence from 1 to 2 degrees in extent.

Latent convergence of more than 5 or 6 degrees is uncommon. Latent divergence of from 1 to 4 degrees is fairly common; more than this is uncommon, but cases of about 12 degrees turn up from time to time.

Hyperphoria of ½ to 1 degree is quite common, of from 1 to 6 degrees by no means uncommon, while there are just a few cases in which from 10 to 20 degrees are to be found. To define my meaning, I may say that I exclude from the above category all cases where there is any history of recent accident or paresis, or where the deviation of the axes of the eyes varies in different positions of the eyeballs, as, on looking up or down, right or left, as it does where one or more of the ocular muscles is not working properly, or where fusion of the images of the two eyes is not commonly attained and maintained, though not without trouble and occasional lapses.

My custom is to examine every private patient, except emergency ones, with the Maddox rod, and if I find any deviation of axis I measure it with prisms, taking the last prism which fails to correct as a measure, and deal with it as follows:

Patients seem to be comfortable if errors of undue convergence are left untreated.

Divergence.

In all these cases the power of convergence must be estimated. Take a prism of about 10 degrees and insert it base downwards in a trial spectacle frame in front of one eye, and, keeping the lamp burning at the bottom of the visual test board at 6 yards, ask the patient how many lights there are. Move the prism a little, if only one is seen, there is quite probably suppression of one image—that is, the patient has monocular vision, using each eye alternately.

If and when two images are seen one above the other, rotate the prism until its base is outward and the patient sees the two images on the same level. If the two images fuse, well and good; there is ample power of convergence. If the two images do not fuse reduce the strength of the

prisms to 9, 8, 7, etc., quickly, so as not to allow the images to fuse when the patient is without a prism, and go on until the images do fuse.

If the patient cannot fuse the images against a prism of 8 degrees base out, convergence is defective and must be assisted.

This can be done by ordering prisms base in, for near work only, of such a strength as to enable the patient to fuse against an 8 prism base out, *i.e.* if the patient could only fuse a prism of 2 base out, order a 3 prism base in each eye. If the error is a big enough one to require a 6 prism base in each eye the weight of the glass is a considerable item and the skin of the patient's nose is likely to suffer, so that a tenotomy of one external rectus must be considered: this gives from 10 to 12 degrees less of divergence or of extra convergence. Muscle training exercises are also useful.

Hyperphoria.

Here errors of a half degree or less may usually be ignored. When the error is greater it should be measured again by means of red and green glasses and corresponding lights. If the error disappears under this test it may usually be ignored; if it persists, try the patient with the correcting prism shown by the later test and the correction for any error of refraction, and ascertain if the prism is tolerated; if so, order it; if not, reduce the strength of the prism till it is tolerated and then order it. If the prism is less than 2, it may be ordered base up in one eye, which is more comfortable than a prism base down for reading; if more than 2, it should be divided between the two eyes.

In some cases the power of fusing against prisms base up and base down respectively will give useful information. For high degrees of hyperphoria, the question of lengthening one or both of the recti concerned will come in, but though the immediate effect is fairly good, from 8 to 10 degrees being gained, the gain is largely transient and the end-result barely justifies the operation.

I find that where a hyperphoria is partly corrected by a prism, which gives immediate comfort, the patient will return about every two years and require a stronger prism to give relief, until nearly the whole original error is corrected, but I do not find that the total error becomes appreciably larger; on the other hand I often, to my surprise, find the power of convergence increase where prisms have been worn for near work only, though no exercises have been systematically carried out.

Acquired Hyperphoria.

This is a rare condition, of which I have seen three cases. The first, a Mrs. T—, whom I saw in 1911, had 6 prism base down left when I first saw her in April, when she also had slight blurring of her optic discs but no true optic neuritis. In May there was no change in the amount of the prism. In June the hyperphoria had changed and the prism had to be reversed to 7 base up left.

In September the prism was 1 base up left. Shortly after this she died suddenly. No P.M. was allowed.

She probably had an intracranial tumour, secondary to uterine neoplasm. Hysterectomy had been performed a year or two before I saw her.

The next case, a man of the middle forties, had intense headache, optic neuritis was well marked, and a marked hyperphoria which varied in degree. He died, and an intracranial tumour was found in his Sylvian fissure. In 1903 he had ½ degree of hyperphoria. In April, 1912, his hyperphoria was 2 base down left; in October, 1913, 6 base up left; in December, 1913, 8 base up left; in April, 1914, 8 base up left. In September, 1914, there was no hyperphoria, he died soon after this.

The third case, a soldier who had been trephined in France for g.s.w., had a marked hyperphoria, but no optic neuritis. I suspected an intracranial lesion. The skull was reopened and six yards of gauze removed, after which the hyperphoria disappeared.

In another case I found hyperphoria of recent onset

in an ex-South African constable; he had also Argyll-Robertson pupils and a positive W.R. In May, 1912, he had 2 prism base down left; in August, 1912, he had 5 prism base down left; in November, 1912, he had 3 prism base down left; in May, 1917, he had 2 base down left.

Under energetic treatment, including heavy inunction of mercury by a masseur, he improved so much that he served in East Africa as an officer and stood the campaign there well.

Squints.

Concomitant.—Why do these occur? Why are they often not noticed until children are several years old?

Newly born children do not use their two eyes together until they are about twelve months old. The tracts in the brain which connect the higher centre, probably in the frontal lobe, with the lower centres, do not receive their medullary sheaths till then, and are not functionally active until they do. In cases of squint something prevents the fusing of the images of the two eyes; what is it? I should say—

(1) Dissimilarity of images, *i.e.* one eye differing markedly from the other in error of refraction, one being nearly normal, and one either highly myopic or hypermetropic.

(2) Marked hypermetropia with over accommodation and over convergence.

(3) In cases with a small amount of hypermetropia or myopia, equal, or nearly so in each eye, and with no amblyopia, hyperphoria may cause it.

One man, æt. 40 years, saw $\frac{6}{5}$ with each eye with a correcting glass, but had over 40 degrees of convergence. After operation and correction of the remaining hyperphoria he acquired stereoscopic vision after exercise.

In another case, a girl, æt. 10 years, who was first

operated on in August, 1914, acquired good stereoscopic vision after stereoscopic exercise. She stopped these and returned in 1919, with a recurrent convergent squint. After another operation in January, 1920, she recovered stereoscopic vision and has kept it, so that even the gaining of stereoscopic vision does not always mean permanent cure.

I believe that the obvious hyperphoria one often sees with a converging squint is the cause of the convergence more frequently than it is caused by the convergence; other muscle errors may also act similarly.

(4) Occlusion: Any temporary covering of one eye at an early age may possibly lead to want of further development or even retrogression, the development of squint and amblyopia, but I am by no means convinced of this.

One boy, æt. 7 years, cut his lids at the inner canthus with a broken plate. Apparently his eyes were then straight and his conjunctiva was not injured. I stitched his lids and bandaged one eye; that eye developed a convergent squint and amblyopia. Curiously enough his father has a slightly amblyopic left eye too. I suspect that I covered a somewhat amblyopic eye, which promptly converged.

(5) Mothers often say their children had straight eyes until they had mumps or measles or any of the exanthemata, and since then one eye has turned. I believe the explanation to be—

There was no true binocular vision and a very slight deviation of axis. This became much more marked when the children were ill, as squints often do, and was then noticed for the first time.

(6) Heredity: There is a strong hereditary tendency to squint; frequently one notices that the mother of squinting children squints too.

So much for causation, now for treatment.

First discover the cause and try to correct it, *i.e.* correct all errors of refraction, hyperphoria, etc., educate the squinting eye to prevent amblyopia, by covering the

sound one for one hour a day to start with, and increase until if necessary the sound eye is only free on Sundays.

If you begin this treatment early in the patient's life you will have a fair chance of success—the earlier the better. Even in the second year after birth begin occluding carefully, even if you do not order glasses until the third year; you will then have fewer amblyopic eyes than if you wait.

After the sixth year your percentage of successes against amblyopia will be smaller and smaller as the patient grows older, so that the first thing to do is to try to prevent amblyopia; in doing so a certain percentage of deviation of axis will be relieved.

If and when a squint in a child becomes truly alternating with a deviation of 20 degrees or more, do not wait too long before you operate, or it may lose the power of fixing with one eye.

As soon as they are old enough to understand the test, try them for hyperphoria and correct it if necessary. Stereoscopic exercises are distinctly useful among the more intelligent.

So, to sum up:

(1) Try to get good vision in each eye.

(2) Try to get fusion of images.

Remember a good many squints disappear in adult life.

About operating: I find a simple and successful plan is to make a conjunctival incision parallel to the margin of the cornea and a little on the corneal side of the insertion of the tendon. Expose the muscle freely. If you wish to shorten it, put a fine catgut suture through it, some little distance back at right angles to its long axis, and then again shortly behind it, so as to get a firm double hold; then pass a needle with the upper end of the suture through the tendon and sclera at its insertion near its upper border, and do the same with the other end; pull tight and knot firmly with a surgeon's knot, leaving the muscle folded. Close the conjunctiva with two or three fine catgut sutures.

If you want to lengthen the tendon, pass the catgut sutures in the same way through the muscle but not so far back. Cut the tendon near its insertion, pass the sutures on a needle through the cut attachment, and allow the tendon to run back as far as you think fit; knot the sutures, cut off the ends and close the conjunctiva as before.

I find 000 catgut causes very little reaction, can be safely buried without resurrecting, and is soft in the conjunctiva and does not irritate. This is a modification of Williams' operation.

Very strict asepsis is essential. To obtain the best results I douche the conjunctiva with 1–1000 acri-flavin, paint the skin of the lids and wash the eyelashes with brilliant green ½ per cent., methyl violet ½ per cent., spir. vini rect. 35 per cent., aq. 64 per cent. This is efficient and not irritating even to the conjunctiva, on which I have often used it. It was recommended to me by Prof. Phillips, of the Jessop Hospital for Women. I cover the skin of the face with a small sterile mask with a window in it.

Paralytic squints.—A very large percentage of these have a positive W.R., and a good many die within two or three years of pachymeningitis, etc. Some paralytic squints are due to encephalitis lethargica, and one must keep one's eyes open for children with squints due to this, as the treatment is certainly not surgical.

Meeting held at Bradford, December 7th, 1921.

3. *A case of corneal tattooing.*

By Charles Killick.

The above refers to the case of Sister A. E—, whose work lies in the V.D. Department of the Bradford Royal Infirmary. She was passing a catheter on a child on May 14th, 1920, when the instrument slipped, splashing some liquid into her left eye. She washed her face immediately and felt no ill-effect until the evening of

the following day, when the eye began to feel sore. The next day following she reported the fact to the matron, who asked Dr. McCulloch to see her, and the latter at once arranged for her admittance into the Royal Eye and Ear Hospital as she was then clearly suffering from gonorrhœal ophthalmia.

She presented a typical picture of the disease: great swelling of lids, chemosis of conjunctiva and after a little while corneal ulceration. The usual treatment was given —silver nitrate, argyrol drops, 1/1500 oily solution of proflavine drops, fomentations, atropine, etc. Antitoxin was also given on several occasions, and, of course, the sound eye was protected by a Buller's shield. I noted on June 3rd that the eye was acutely inflamed and the cornea deeply ulcerated. Vision was P.L. On July 27th the case-paper reports a small corneal fistula.

On August 16th, 1920, which would be some time after she had left the hospital, I again examined the eye. It had now recovered from the inflammation, but the corneal surface was covered for almost its entire area with a dense white opaque scar; only a very small portion situated near the inner margin retaining a degree of transparency. The eye was hideously disfigured and the patient felt this very acutely, and although she returned to her work in the V.D. Department, she practically ceased visiting her friends and so on. Fortunately, the right eye was saved and was nearly normal: vision was $\frac{6}{6}$ with a weak astigmatic glass.

I next saw Sister E— on October 5th of the present year. The ocular condition was the same. The leucoma appeared to be of uniform density except at one spot, where it seemed to be rather thinner and darker; probably this was the site of the corneal fistula mentioned above. There was no staphyloma and the surface was white and even. As she was very anxious to have something done to mitigate the deformity I arranged to tattoo it. The patient was admitted and the following day was operated upon under cocaine anæsthesia. Thick China ink was

used. A little of this after being sterilised by heat and diluted with a little sterile water was poured into a watch-glass, and then by means of an ordinary tattooing needle multiple superficial punctures were made all over the corneal surface, which was quickly hidden under a small pool of ink. The effect of the tattooing was observed every minute or so by washing off the excess of ink with boracic lotion. I was, of course, particularly careful to be very gentle when tattooing near the apparently weaker part of the leucoma. Having done what I considered sufficient for one sitting the operation was discontinued and a dry dressing applied. The patient had hardly any pain either during or after the operation, and scarcely any reaction followed, so that she was able to return to work two days afterwards very much delighted with the effect already produced, and which was indeed sufficiently striking.

On October 8th she came up for another sitting, and, as it proved, the last. The procedure was repeated, the tattooed area was carefully gone over again, and any little white spot which had escaped on the previous occasion was well punctured. She felt this operation a little more and there was a little more reaction afterwards, but no iritis fortunately set in, and she was discharged three days later. I saw her once a day or two afterwards but have not seen her since then. You have been able to see for yourselves this afternoon the really wonderful improvement which has been effected. I have seen it stated that the small punctures made in the conjunctiva by the fixation forceps may themselves be tattooed by wandering drops of ink, and that therefore forceps with ivory or rubber ends should be employed. In the two cases I have done since coming to Bradford the eye was fixed in the usual way without any untoward result.

As you are all aware the operation of tattooing dates from antiquity. Galen touched the leucoma with the end of a red-hot probe, and then applied a mixture of gall nuts or pomegranate seeds mixed with a salt of copper. The

operation was re-introduced by de Wecker and Abadie. Not every eye, however, lends itself to tattooing. There are two main contra-indications: the scar must not be too recent, or too rich in vessels, and the iris must not be extensively adherent. If the iris is much involved in the scar the operation is likely to be followed by irido-cyclitis, and even, according to Terrien, by sympathetic ophthalmitis.

Other pigments besides ink have been used. Rosselli has employed the pigment from the eye of a crow. More elaborate still, the pupil has first been delineated in China ink in the ordinary way and the rest of the cornea tattooed with appropriate colours to match the good eye.

The only alternative to tattooing short of total removal of the eye is either partial or complete corneal grafting, or kerato-plasty. In the case under review it was, of course, unnecessary to consider it as the right eye was sound and healthy. Grafting has, however, proved very disappointing: it is easy to get the graft to take, but it only in the rarest instances remains transparent. I have done successful grafting twice but in neither case did it remain transparent.

It is important that the whole thickness of the cornea of the eye which is to receive the graft should not be removed, but that Descemet's membrane should be respected. Homoplasty is best, and Magitot has shown that excised eyes will retain their transparency when placed in hæmolysed serum for about twelve days. Transplantation from a young rabbit's eye is always attended with failure, as the graft merely serves as a scaffolding for the reparative process and is replaced in time by opaque tissue. Moreover, the graft must not exceed 4 mm. in size or it will not take.

I merely mention grafting as it is rather a fascinating subject and has attractions for the young surgeon. It is, however, hardly a practical proposition.

Considering the number of leucomata which are met with I think the operation of tattooing might be done

more frequently than it is, and I have therefore brought this case to your notice. The result from the cosmetic point of view you will agree has been extremely satisfactory.

REFERENCE.

TERRIEN, F.—*Chirurgie de l'œil.*

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4. *Some remarks on ophthalmoplegic migraine.*

By GEORGE H. OLIVER.

WHEN asked by our Secretary to contribute something towards the proceedings of this meeting I truly felt at a loss what to say, and at a still greater loss if I did say anything what it should be about. At meetings of this character where all the members are specialists, it is difficult to say anything on any branch of ophthalmology which is not well known to all. I think the chief interest of these meetings is obtained from the exhibition of cases of unusual character. I am sorry to say that for the first seven months of this year I did no work on account of illness, and as a consequence lost the opportunity of looking out for interesting cases during that period, and my colleague tells me that as far as his experience goes there has been a singular absence of cases which are out of the ordinary run of an eye clinic. In choosing a subject on which to speak, one is driven to select one which is of more than usual interest, examples of which are perhaps not very frequently seen in the practice of any of us. I have therefore been persuaded in my own mind that a few remarks on ophthalmoplegic migraine or relapsing oculo-motor paralysis might not be out of place. I have seen two of these cases: one many years ago which occurred in a patient under one of my colleagues at this hospital; the other was my own patient, also at this hospital some years ago. Of the former case I remember very little and have altogether failed to trace the records.

The other—my own case—was a woman past the menopause who presented herself as an out-patient with a complete oculo-motor paralysis, internal and external. She said she began with this peculiar condition early in life—pain in the head, vomiting and paralysis of eye muscles, ptosis and diplopia being the causes of most of her trouble ; after the pain had passed off, the symptoms caused by the paralysis disappeared so rapidly that she did not seek advice. She had several subsequent attacks in which pain was of longer duration followed by increasingly lengthened periods of paralysis, until she got into the condition which a high proportion of these cases experience, viz. complete permanent oculo-motor paralysis, and it was owing to the continuance of the paralysis that she sought advice. I saw her a few times, but there was no improvement in her condition, which I took to be permanent.

Like many other ophthalmic surgeons I felt very much interested in this kind of case, and, like others, wondered what could be their nature. In the first place they are comparatively rare, many men not having seen a case through a long experience. It is very remarkable that patients suffering from this disease are invariably of the poorer class and are usually discovered among hospital patients, whilst those who suffer from ordinary migraine are by no means confined to that class. These cases are so rare that Fuchs in his text-book dismisses the subject in two or three lines. Römer says, "The disease is of extraordinary interest to ophthalmic surgeons and neurologists, and a symptom-complex of such rarity, as a typical case presents, justifies and is worthy of careful study ; moreover, as to the nature and true cause of the disease we are in complete darkness." This peculiar disease received the name recurrent oculo-motor paralysis from Charcot. Remak, Senator and Vissering considered it a functional condition—hysterical—a hysterical reflex paralysis, and considered it justifiable so to name it, until more light was shed on such an obscure condition.

Mœbius, who was the first to describe this complaint in its entirety, understands by relapsing oculo-motor paralysis those cases in which, from youth or early childhood, paralysis is limited to the third nerve, commencing with headache and vomiting, returns at longer or shorter intervals, and affects nearly always one and the same eye. In typical cases the disease occurs in three periods—first, pain and vomiting, second, paralysis, and third, intervals between the attacks.

Period of pain.—The pain usually begins in the temporal region and spreads to the occiput, accompanied by nausea. Mœbius says the pain is not a true neuralgia, in that it does not occur in distinct spasmodic attacks, nor does it affect single nerves; it is a diffuse pain such as is experienced by patients suffering from migraine or cerebral tumour, and is accompanied by vomiting as in migraine and tumour, which is not the case in the most acute neuralgia. It is characteristic that the pain and vomiting precede the paralysis, and these cease as soon as the paralysis is fully developed. These symptoms show that the paralysis is not of peripheral origin. A lesion affecting the trunk of the third nerve, if it is painful, must cause a simultaneous irritation of the sensory branches of the fifth nerve or local irritation of the dura mater, and disturbances of sensation have been demonstrated by several observers, such as tactile and thermal hyperæsthesia of the first division of the fifth nerve, paræsthesia of the nostril and painful pressure over branches of the fifth nerve—all on the side of the affected eye. In some cases ordinary migraine is followed by ophthalmoplegic migraine as recorded by Charcot, Manz, Joachim and others; in some ordinary migraine alternated with ophthalmoplegic migraine, as reported by Snell, Suckling and Senator, and in some cases patients could tell by the character of the pain whether ordinary migraine or ophthalmoplegic migraine was in process. Mœbius thinks the pain has its origin in the nuclear region, as the cell groups of the origin of the fifth nerve lie to the outer side

and above the third nerve group, and as these (fifth nerve) supply the eye and dura mater with sensibility, it is easily understood that an œdema affecting the third nerve origin could easily affect the fifth nerve cells and thus produce headache and reflex vomiting. The acute stage over, swelling subsides, irritation ceases and only paralysis remains. The pain is practically always on the same side as the oculo-motor paralysis. The length of the painful period is very variable—from two days to two weeks or even two months. Schmidt-Rimpler records two cases where headache was absent. The first sign of the onset in a case of Snell's was a sinking of the left upper lid—the migraine occurring on the following day. Occasionally vomiting precedes the headache.

Period of Paralysis.

This commences as soon as the pain has ceased and occasionally during the pain, so that pain and paralysis co-exist. In Schmidt-Rimpler's case the first attack was unaccompanied by pain. The paralysis sometimes commences suddenly; usually it develops gradually and is complete in a few days. Mœbius, Saundby, Ormerod and Spicer, Thomsen and others record cases in which the paralysis was at once complete. In other cases paralysis in the first attack is incomplete and has become total in later attacks. Other records show that almost any muscle supplied by the third nerve may escape, the iris may be paralysed and accommodation escape. Some muscles may be slightly affected, while others, though supplied by the same nerve, may be completely paralysed. The duration of the paralysis is of great variation in the individual as well as between separate cases. In one case the lid drooped for one day only. Mydriasis and paralysis of accommodation are the last to clear up, and occasionally persist during the interval between attacks (Clark, Snell and others).

It is interesting that in some cases the paralysis has recovered with the exception of the levator, ptosis

persisting. Karplus records a case in which a patient six months old had an attack of vomiting followed by right-sided ptosis, which disappeared in a few days. Vomiting attacks occurred every four weeks up to twenty years. Later there was mydriasis and external strabismus, which continued between the attacks. Post-mortem a tumour the size of a split-pea was found in the trunk of the third nerve—a neuro-fibroma, which completely infiltrated the nerve, the fibres of which it separated.

Period between the Attacks.

This may be divided into two groups: (1) In which paralysis disappears entirely; (2) those in which one or more muscles remain paralysed. The great majority of cases belong to the latter group. The duration of the interval between the attacks, as said before, varies greatly. Several different pathological conditions have been found to accompany oculo-motor paralysis, viz. paralysis of muscles supplied by other nerves, hyperæsthesia in distribution of first and second division of fifth nerve, anæsthesia of cornea, atrophy of optic nerve, choroiditis in the macular region, lowering of visual activity, scintillating scotoma, concentric contraction of visual field, tinnitus and deafness, excessive sweating, increased nasal secretion and salivation. Regarding the ætiology and nature of relapsing oculo-motor paralysis, its near relation to migraine may be referred to. Many authors have recorded cases in which patients have for years suffered from migraine of the usual type, which has been followed by ophthalmoplegic migraine, and these attacks sometimes alternate, as has been referred to, and although both conditions stand in near relation to each other, patients who suffered from both had no difficulty in saying which was present in any given attack. The persistence of paralysis between attacks shows that it is not a true migraine, particularly as the attacks do not occur so frequently as in that disease, *i. e.* weekly or monthly, but more commonly two to six times in a lifetime, and continue for days—indeed sometimes for

many months. Several authors have expressed their opinion as to the nature of the disease. These I have not time to go into, but it suffices to say that of all the given explanations none is satisfactory. Senator thinks that those attacks which are followed by complete disappearance of the paralysis should be considered purely functional, and those in which paralysis persisted, organic. Mingazzini thinks that the free periods are characteristic of the early stage, and disease is not fully developed until paralysis is permanent. Against this Charcot recorded a case in which after the last attack paralysis disappeared entirely. Oppenheim thinks that as we get cases of migraine without paresis and hemianopsia, so we can get migraine without oculo-motor paralysis. Massalango thinks that in most cases inflammatory processes or new growths in the nerve are at the root of the mischief. Marina thinks that the disease is one essentially of peripheral neuritis of the third nerve. The most we can say is that this disease is not to be considered as one in itself, but as a symptom-complex, which from variable causes fastens direct on to the third nerve, and whose manifestations are the same in most cases. The real cause is not known. It begins nearly always in early life, may commence later even in the sixth decade, but the greater the age the less the case incidence. Both sexes are affected in approximately equal numbers. Of the course of the disease so much may be said, that in most cases finally more or less complete oculo-motor paralysis remains, and, sometimes, between the attacks. Diagnosis is not difficult, as appearances are characteristic, and the characteristic symptoms have been mentioned, though some conditions might give rise to the suspicion that we were dealing with ophthalmoplegic migraine when post-mortem examination revealed the cause of the symptoms, which were not really characteristic of our subject. This is the case in syphilis, tabes and chronic meningitis. One case is said to have been of malarial origin, as it was always cured by quinine. It may be simulated by hysteria, but other symptoms serve to

show the real nature of the attack. The numerous causes of recurrent paralysis of the third nerve show how difficult the diagnosis may be. Among others the following should be excluded: Intra-cranial tumour, aneurysm of basal vessels of the brain, swelling of the cavernous sinus, cerebral syphilis, tabes, syringomyelia, chronic meningitis, multiple neuritis, hysteria, and multiple sclerosis. All these must be excluded before the diagnosis of ophthalmoplegic migraine can be made. It is not at all unlikely that increased experience will throw more light on this mysterious disease. Only two post-mortems have been made, in which the opportunity was given to make an examination of the affected parts. It seems to me that the symptoms may be produced by a toxin, especially the paralysis, and if this toxin causes a local œdema, which is supposed by Mœbius to be the case, the pain and reflex vomiting would be accounted for.

So far as treatment is concerned, Römer says—"This is purely symptomatic."

Meeting held at Liverpool, February 10th, 1922.

5. *Notes on malarial conditions of the eyes.*

By H. Haward Bywater.

Synopsis.

A. Frequency.
B. Period at which they occur, and in what form.
(1) Corneal conditions.
(2) Hæmorrhages.
(3) Uveal affections.
(4) Muscular paralyses.
(5) Conditions possibly malarial.
(6) Other recorded conditions.

How many of these conditions can be considered true complications of malaria?

The following notes on malarial eye conditions are based on personal experiences as Eye Specialist to H.M. Forces in Salonika in 1917 (with Major C. H. Usher, of Aberdeen, as my chief), and on cases seen in ex-service men since.

A. Frequency.

Eye complications have been said by Poncet to occur in 10 per cent. of malarial cases. This estimate, according to Elliot, is a grave exaggeration. It has been difficult to estimate their frequency, largely because (*a*) the ophthalmologist sees little of patients in the acute stage of malaria, (*b*) because the percentage of malignant cases (which cases are chiefly responsible for fundus complications) is in India much smaller than in Macedonia and in Africa, and the estimates have often been based on Indian experiences.

B. Period at which they Occur, and in what Form.

Superficial corneal conditions: Occur in the early days of the malarial attack, and are associated with benign malaria.

Iritis choroiditis: In malarial cachexia. My two cases of choroiditis were associated with benign malaria.

Retinal hæmorrhages: (*a*) Small and peripheral during pyrexial attacks. More often in malignant than benign. (*b*) Large and central in malarial cachexia, especially if anæmia is profound. In malignant malaria.

Optic neuritis: Generally during convalescence. Also has been found post mortem in two cases of malignant malaria (Poncet).

Muscular paralyses: About the end of the third week.

In malarial cases we get a slightly yellowish tinge in the conjunctivæ due to hæmosiderin.

In the chronic cases the eyes are sunken and dull, and the skin and mucous membranes generally are more or less pigmented, due to the black pigment hæmozoin.

I have divided this short article into parts:

(1) Corneal conditions.

(2) Hæmorrhages—of conjunctiva, vitreous, and retina.
(3) Uveal conditions—iritis, choroiditis.
(4) Muscular paralyses.
(5) Conditions observed which were possibly malarial.
(6) Other conditions recorded by various observers.

(1) The Cornea.

(1) The commonest complication is dendritic ulceration. This is accompanied by anæsthesia of the cornea, and occasionally of the eyelids and surrounding skin, and also often by herpes of the face, especially of the lips, the angle of the mouth, and the side of the nose.

These ulcers commence by the fusion of small round infiltrations, which, after coalescing, take various forms (stellate, racemose, spirillar, linear, V-shaped, etc.). They are raised from the surface, and at first stain feebly, if at all, with fluorescein. Next a depression or rill appears along the infiltration. This stains brightly with fluorescein. At the extremities of the ulcer are recent infiltrations which scarcely stain. The infiltrations are usually grey, but in some cases vesicles may be seen.

(2) More rarely, another superficial corneal condition, a patch of vesicles surrounded by infiltrations *with no ulceration,* may be seen.

Onset.—These superficial ulcers usually appeared on the second to the fourth day after the onset of the attack. Occasionally they preceded it ; sometimes they appeared as long as three weeks after the attack.

Complications.—(1) Iritis is very rare. I only saw it in one case.

(2) Hypopyon ulcer: Two cases became infected and were accompanied by a mild hypopyon.

Duration.—The duration was about four weeks.

Cultures of scrapings from the ulcers were made in three of the cases with negative results.

Treatment.—Simple treatment. Guttæ atropinæ with

boracic lotion along with the constitutional treatment of malaria gave the best results. The cautery is contra-indicated.

Quinine drops were used in some cases without appreciable benefit.

Out of 92 cases of corneal trouble of which I have notes 64 were associated with malaria, 60 of these being dendritic and 4 not dendritic.

In 9 other cases dendritic ulceration occurred where malaria was not proved.

Though this form of keratitis is met with in other conditions of fever and debility, as after influenza, it is more often seen in association with malaria than with any other illness. (Pathology unknown. Probably a toxic condition of the peripheral nerve-endings; possibly also of the sensory portion of the ciliary ganglion.)

(2) Hæmorrhages.

(1) *Subconjunctival ecchymoses* near the limbus have been seen at the commencement or during an attack. I do not think they have been previously described.

(2) *Vitreous opacities* are also occasionally seen shortly after a malarial attack, and are usually associated with retinal hæmorrhages.

(3) *Retinal hæmorrhages* occur chiefly in the malignant forms of malaria, and are (*a*) *subhyaloid*, having the usual shape of the lower half of a circle. Occasionally they are pear-shaped or irregular. Later when undergoing absorption they may become crescentic. After absorption small white dots are often seen in their site. These subhyaloid hæmorrhages are usually in the macular region or just below it. In some of the cases the vision becomes quite normal, but in many is permanently deteriorated. (*b*) *Flame-shaped hæmorrhages* often occur in conjunction with the subhyaloid hæmorrhage, or alone.

These retinal hæmorrhages were seen almost invariably in the *subtertian or malignant* form, and often in cases where there was marked *anæmia*. In one case hæmo-

globinuria in a severe form occurred after quinine had been given in large doses; at the same time there was a second very severe retinal hæmorrhage.

Embolism of the Retinal Artery.

In one case of malignant malaria the right retina was greyish and hazy, especially on the nasal side near the disc; the retinal artery below was much narrowed, and the veins unusually dark. The macula was more red and dark than usual, but there was no cherry-red spot. The vision in this eye was only hand-movements, and the appearances resembled those seen in embolism of the retinal artery.

(3) Uveal Affections.

Iritis.—I have notes of 6 cases of mild iritis. I found it difficult to exclude absolutely all other causes of iritis. Major Smith, however, records 15 to 20 cases of iritis of a mild type, where he could exclude other causes; these cases became worse for the time with each rise of temperature, and were amenable to local treatment plus quinine.

Choroido-retinitis.—In two cases I have seen following *benign malaria* a superficial retino-choroiditis affecting both eyes, yellowish-white patches of various sizes extending over part or the whole of the fundus.

These in places coalesce, leaving darker islands between. There is moderate pigmentation in small patches. A few larger pigment spots were seen in the periphery, the disc and retinal vessels being practically normal. In this condition the vision was much better than the fundus changes might lead one to expect, but there was some night-blindness.

In one of these cases a diminution of the visual acuity occurred at each recurrence of malaria.

There was no specific history in these cases, and a Wassermann was negative in each case.

Yarr states that an œdematous state of retina, disc and

fundus generally is seen in 20 per cent. of acute intermittents.

This is the first stage of malarial retino-choroiditis and may subside quickly.

(4) Muscular Paralyses.

Cases met with were—

Paralysis of external rectus.—I have seen one case (Kirk states that the sixth nerve is more often involved than the third or fourth).

Paresis of internal rectus.

Complete or partial paralysis of third nerve.—In the second case shortly after an attack of malaria the right eye began to diverge, though no double vision was manifest; the patient preferred to keep a pad over it, the eye did not turn fully to the left, and there were nystagmoid jerks.

Complete third nerve paralysis.—In this case sudden pain in one side of the forehead came on nearly three weeks after his first attack of malaria; the next day there was complete ptosis on that side. There was no specific history, and a Wassermann test was negative. When seen three weeks after there was complete paralysis of the third nerve and the pupil was dilated. The fourth and sixth nerves were not affected; no other cranial nerves were affected. These paralyses often improve very slowly. I have recently seen a case where there was history of complete third nerve paralysis following malaria in 1916.

At the present time there is still paralysis of the right inferior and internal rectus. The ptosis has disappeared and the pupil is normal.

(5) Doubtful Cases.

Certain conditions such as an attack of tenonitis and two cases of detachment of the retina in otherwise normal eyes were suggestive of malaria. These may have been due to hæmorrhages.

(6) Other Recorded Conditions.

Many other conditions have been recorded by different observers.

The cornea.—Diffuse interstitial keratitis: these usually showed typical clinical signs of congenital syphilis and gave positive Wassermann tests. Malaria may have precipitated the attacks. Keratitis profunda, or deep-seated keratitis: a central circular discoid infiltration in the deeper layers of the cornea (Arlt, Levrier, Maxwell).

The sclera.—Periodic transient episcleritis is mentioned by Fuchs. Maxwell reports two cases of episcleritis, one giving a positive Wassermann test, and neither responding to quinine.

Lens.—Cataract has been noticed. Four cases in young cachectics were reported by Krachly (Bourland), and 4 by Bagot (Yarr).

Vitreous.—White infiltration of the vitreous, rare, was first described by Seely (Yarr). It consists in an infiltration of the vitreous forming in stages, causing almost complete loss of vision for a time and giving a characteristic white reflex with reflected light. Seely attributes it to a serous infiltration due to chronic paludism.

Optic neuritis.—Poncet, Yarr, Bourland and Maxwell have described several cases. They usually appear during convalescence.

The papilla is said to be of a dirty reddish-grey tint due to parasites in the capillaries. Yarr states, "The variations in the visual acuity in the course of the malady form the most characteristic symptom and distinguish malarial neuritis from all other forms."

Regarding the pathology of this, Yarr says, "Poncet shows conclusively that the changes in disc and retina are due primarily to melanæmia with increased vascularisation, the subsequent atrophy or partial atrophy being explained by consecutive endarteritis of vessels. In his opinion 80 per cent. of cases terminate in a partial atrophy."

Retrobulbar optic neuritis.—Kirk has described one case.

Optic atrophy may follow the optic neuritis, or may be due to quinine.

Retinitis.—Poncet has described an albuminuric retinitis in association with nephritic malaria.

Retinal hæmorrhages. — Besides the larger retinal hæmorrhages at the posterior pole similar to those I have described, minute peripheral punctate retinal hæmorrhages, occurring in the febrile stages, and said to be much more common, have been described by Poncet, who found them in all cases of death from malaria. They are so minute and so far forward as to be often overlooked.

Retino-choroiditis.—Yarr states that 20 per cent. of acute intermittents complain of supraorbital pain and photophobia, and that these show an œdematous state of retina, disc, and fundus generally. The retina appears to have an undulating surface, dunes and intervening depressions. This is the first stage of malarial retino-choroiditis, but may subside quickly.

Finally one or two obscure conditions may be noted:

Sudden and persistent amaurosis (*a*) without visible fundus change have occurred. These are due to some obscure focal brain lesion.

(*b*) Periodic amaurosis. May be due to œdema of retina, or hæmorrhages, or to quinine.

(*c*) Sudden amaurosis ending in atrophy. Possibly due to hæmorrhage into sheath of optic nerve; occasionally due to quinine.

(*d*) Persistent central scotoma. Always due to macular hæmorrhage in the experience of Yarr.

(*e*) Periodic blue vision (Baas); cause?

We see many of these conditions occurring apart from malaria, and it is a natural question to ask, How many of these conditions may be really due to the malarial parasite? We know that some eminent authorities who have done years of work in tropical countries, but have had experience chiefly of benign malaria, agree that the superficial corneal conditions may be due to this, but have little to say of the fundus complications. Among

those who have seen much of malignant malaria there is no doubt at all that these fundus conditions occur. Unfortunately few statistics are available.

One thing that impressed us was that whilst we got comparatively many eye complications from our cases of malaria, we got scarcely any at all from an equal number of dysentery cases, seen at the same time.

Dealing with the different conditions in turn :

1. *The superficial corneal conditions*—dendritic ulcers—occur in other feverish conditions. Uhltoff considers 25 per cent. of all corneal ulcers seen in army service were of this type. No parasites have been found in scrapings from the ulcers. On the other hand Kipp found 9 out of every 10 of his cases were malarial. Elliott states: "This has been so widely recognised as a malarial manifestation that we may probably accept it with but little hesitation; on no occasion has the malarial parasite been found in scrapings from the cornea. This of course does not exclude the possibility that the affection may be due to the action of a specific malarial toxin." Periodicity appeared to be sometimes present, a recurrence of the fever being accompanied by a fresh corneal invasion. The herpes of the face which often accompanies these ulcers is accepted by Castellani and Chalmers as a benign malarial complication.

It is doubtful if there is any reliable evidence that a true *interstitial keratitis* is ever caused by malaria.

Keratitis profunda.—Elliott appears to be doubtful about this. He says, "Few worse examples can be found of the evil method of copying which disfigures the majority of text-books."

Iritis and choroiditis are met with in malarial patients just as they are in non-malarial. Major Edgar Smith's cases (he met with 15 to 20 cases of low-grade iritis readily amenable to local treatment combined with quinine, which showed exacerbations corresponding to rises of temperature; blood tests were positive to malaria, and syphilis, dysentery, gonorrhœa, dental sepsis and digestive

troubles could be excluded) seem to prove that we can have a malarial iritis.

Choroiditis.—The malarial parasite has been found in the larger choroidal vessels (Castellani and Chalmers). In one of my cases where syphilis was excluded there was undoubted periodicity; the vision became worse for a time with each rise of temperature.

Optic neuritis.—Elliott says, "There would appear to be sufficient evidence to include malaria among the diseases which may cause it." The most interesting evidence regarding optic neuritis and retinal hæmorrhages is the record of the post-mortem examination by Poncet in 1878 of the eyes of 2 patients who had died from malignant malaria. Before death he had noticed ophthalmoscopically (1) optic neuritis, swelling of the papilla with a dirty grey tint in the middle zone of the optic nerve; (2) peripapillary œdema; (3) retinal hæmorrhages, punctate at the periphery, and very large at the posterior pole. Histologically he found (*a*) a granulous colourless liquid poured out under the internal limiting membrane of the retina near the papilla; (b) increased vascularisation with interstitial œdema in the disc, the dirty grey tint being due to the great number of white pigmented corpuscles enclosed in the hyperæmic vessels; (c) extravasations of white corpuscles in the choroid, which was more voluminous, irregular and swollen near the papilla, the internal tunic of the vessels being not much affected. He also examined the eyes in a case of malarial nephritis. Here the changes were those of ordinary albuminuric retinitis, but in addition there were large pigmented elements in the capillaries. Elliott says, "The small petechial hæmorrhages we must regard from the pathological standpoint as being identical with like phenomena in other organs of the body."

Malarial cachexia is associated with profound anæmia, and profound anæmia is frequently associated with hæmorrhage in many parts of the body. Major Edgar Smith says he never saw a case with a red cell count over 3 millions.

Muscular paralyses.—In my cases we excluded syphilis. According to Elliott, "There is a wide-spread belief that paralyses of the external muscles of the eye may occur as a manifestation of malaria."

Other eye conditions, as detachment of the retina and cataract, are speculative.

When one considers how profound the blood changes and how intense the toxæmia is in many cases of malaria, one feels that they explain very many of these conditions.

Could *quinine* have anything to do with this? I think not. At any rate an overdose of quinine gives rise to the pale disc, contracted retinal vessels and concentrically contracted fields—quite a different clinical picture.

In conclusion I beg to acknowledge my deep indebtedness to Major Usher, to Lieut.-Colonel Elliott for the references to his book on *Tropical Ophthalmology*, and to other writers.

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6. *Two cases of neuro-fibromatosis.*

By H. Horsman McNabb.

Dorothy M—, æt. 5 years, came under my care at the Manchester Royal Eye Hospital first on November 13th, 1907. The child's mother said the left eye had been prominent for one month. On examination the left eye was found to be displaced forwards. Movement was good in every direction. No tumour could be felt in the orbit, but there was slight resistance on attempting to push the globe backwards. The pupil was normal. The optic disc was congested. V. = Fingers easily.

The eye became more proptosed, and on January 11th, 1908, there was well-marked optic neuritis and V. = Fingers with difficulty. Movement remained good in all directions. On February 14th, 1908, I enucleated the eye, and although the optic nerve was divided as far back as possible, I did not succeed in removing the whole of the tumour, but by pulling forward the stump of the nerve I was able to excise a further small portion and apparently get away the remaining part of the tumour

Some months later a glistening white mass appeared behind the conjunctiva, but although I was able to keep the child under observation for some years this mass did not increase in size.

Fig. 11.

Pathological Report by Prof. Dean.

"Intra-dural neuro-fibromatosis with proliferation of the neuroglia."

Mary B—, æt. 9 years, came under my care at the Manchester Royal Eye Hospital first on September 8th, 1917. Her mother said the right eye had been prominent one month.

On examination the right eye was slightly proptosed; movement was good in all directions. No tumour could be felt in the orbit and there was no resistance on backward pressure of the eye. The pupil was normal. The optic disc and rest of the fundus normal. $V = \frac{6}{6}$, $\bar{c} - 3 = \frac{6}{6}$.

During four years the proptosis gradually increased and a large tumour mass could be felt in the orbit. The optic disc became pale and V. was reduced to $\frac{6}{9}$, J. 19.

On July 13th, 1921, I made an incision along the outer orbital wall, and on examination with the fingers felt a large growth around the optic nerve, and it seemed to me impossible to remove this without sacrificing the eye. I enucleated the eye and cut through the optic nerve as far back as possible. On macroscopic examination the nerve itself was not thickened, but the sheath appeared like a piece of omentum with hard nodular masses scattered throughout it; this was nowhere adherent to the nerve itself.

PATHOLOGICAL REPORT by PROF. DEAN.

"The tumour mass is one of neuro-fibromatosis."

Meeting held at Leeds, March 10th, 1922.

7. *On the physiological night-blindness of the one-eyed.*

By THOMSON HENDERSON (Nottingham).

THE night-blindness of the one-eyed is a subject which, as far as my researches go, has not been noted in ophthalmic literature, and yet it is one of some practical bearing.

Before the war one had met with isolated cases in which one-eyed workers, chiefly miners, had complained of inability to see at night, but personally I had never given this much consideration as I had never met with this disability before the passing of the Workmen's Compensation Act, and I therefore ascribed it as belonging to one of the numerous class of functional manifestations to which this Act has undoubtedly given rise.

During the war this complaint was, however, early and very forcibly brought before my notice in consequence of the large number of men who had lost an eye in action, and who complained of this disability.

These men, all on home service, complained of the same disability—that although perfectly fit and competent to carry out their duties during the day, yet at night they were unable to see, and therefore incapable of doing their turn of sentry duty.

On examination I cannot remember ever having found in the remaining eye fundus changes or appreciable refractive error.

On testing with Snellen's test-types and reducing the illumination in the room their adaptation was every way as good as my own. Thus if the light only allowed me to make out $\frac{6}{18}$, they equally were able to make out the same, and if the illumination was still further diminished, allowing me to see only $\frac{6}{36}$, they likewise unhesitatingly made out the same. Thus anything in the nature of true night-blindness was out of the question.

I admit that I could not understand their complaint, though forced to the conclusion that it was some subtle form of functional condition such as was later popularised under the name of "shell-shock." I therefore always made a recommendation excusing these one-eyed men from night duty under the plea that they had not yet got accustomed to one eyed-life.

The solution of the problem came to me during the last winter of the war when I was temporarily forced to join the ranks of the one-eyed by a carbuncle over the left supra-orbital margin. One evening, after having had the obnoxious excrescence incised, I suddenly discovered that I literally could not see the way back to my quarters. At first I thought the difficulty was due to some fault in my retinal adaptation, though I knew that ordinarily it was really very good. Lifting up the bandage and looking now with my left eye alone I at once appreciated a marked difference, as, owing to retinal adaptation, everything appeared brighter as compared with the original uncovered right eye, but still I failed to distinguish objects any clearer.

With both eyes open, however, I found that I could

make out everything as clearly as one could with the given illumination of a winter evening in war-time, when all sources of artificial light were kept hid under a bushel.

I at once appreciated the fact that the whole cause of one's difficulties was due to the loss of stereoscopic vision. Ordinarily in daylight or with the artificial illumination that our streets now present, the loss of stereoscopic vision does not cause much difficulty, as secondary factors come in to assist monocular vision to appreciate the third dimension.

In the absence of artificial illumination these accessory factors by which monocular vision can appreciate depth are, however, unable to come into play even in moonlight. The result of the complete loss of stereoscopic perception is that objects in the foreground are thrown and merged into the background, producing a perfectly flat monochromatic picture of varying shades of grey and black, in which one is unable to distinguish any object because of the absence of relief. The effect is very disconcerting, as one has the feeling of literally walking up against a black and blank wall.

With binocular vision this dead flat monocular picture resolves itself at once into a stereoscopic one, in which one can recognise objects, as now they stand out in relief, though they may only be seen as black masses.

It is this stereoscopic effect that monocular vision entirely loses at night time and hence the feeling of being up against a blank wall.

You may in a measure appreciate how diminished illumination causes loss of monocular perception of depth by entering a room just after sunset with one eye closed.

You will be able to recognise and distinguish the familiar objects about the room, but darken it still further by pulling down the blinds and you will note how everything literally fades away into the background; open both eyes and the position of everything can be made out again.

Like many another apparently puzzling condition the explanation of the night-blindness of the one-eyed is a very simple one, though it really was the last that one thought of.

IRISH OPHTHALMOLOGICAL SOCIETY.

President :

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W. M. Killen.
H. C. Mooney.
W. McCready.
A. W. Sandford.
L. Werner.

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W. C. MacFetridge.

REPORTS.

Clinical Meeting held at the Royal Victoria Hospital, Dublin, June 8th, 1921.

The following cases and communications were brought forward and discussed :

1. *Case of monocular blindness followed by recovery in a few days.*

By W. M. Killen.

A young lady teacher, æt. about 25 years, came to me in April, 1918, suffering from floating opacities in the vitreous of the left eye. The vision of this eye was $\frac{6}{12}$.

Three years later she returned with this eye in the same condition, but complaining that the right eye, previously perfect, had, some days before, become almost blind, and a day or two after, absolutely blind. I found the pupil widely dilated, with no response to light, although it contracted perfectly to the consensual reflex test. The ophthalmoscope showed the fundus to be apparently normal. The treatment was free leeching of the temple and pilocarpine sweats, etc. In eleven days the vision had gradually returned and was soon $\frac{6}{6}$. We all see occasionally such cases of monocular blindness without obvious lesion clear up with or without treatment. The patients are frequently young women. I offer the case for remarks from the members.

Mr. L. Werner thought there were two explanations: (1) Functional; (2) obstruction of circulation, the blood-pressure low and not enough to drive the blood through the arteries.

2. *A case of endogenous infection after needling for cataract.*

By W. M. Killen.

A few months ago a young woman, æt. 35 years, came to me with a mature cataract in the right eye. The left eye showed incipient cataract.

There was a history of cataract among her relatives, and her parents were cousins.

Believing the lens would prove soft with a very small, if any, nucleus, I needled it with all care as to asepsis. A few hours after this simple little operation, a very acute attack of iritis with hyperæmia of the whole eyeball developed, and threatened the destruction of the eye.

The tension was not much raised. The attack was overcome by vigorous treatment for a fortnight by hot stupes, atropine, leeches, etc. I had a culture made by an expert bacteriologist from the conjunctival secretion,

and the report was that it was sterile. He also made a culture from a catheter specimen of the urine and found a growth of *Staphylococcus albus*, and reported the patient as a staphylococcus carrier. A vaccine was made from this and the patient treated with it for a period of three months, after which, the eyes being quite quiet, the lens was extracted through a small linear keratome incision in the cornea, with the aid of free irrigation, since the cortex was sticky. There was no inflammatory reaction from this more severe operation, and the visual result is fairly good and improving, although there is some striate opacity of the cornea. I give this case as an example of autogenous infection. The question of the best operative procedure for cases of about 35 years of age is also interesting and deserving of discussion, since in Ireland one frequently finds a cataract quite soft, and with little or no nucleus, up to 37 or 38 years of age.

Mr. L. Werner asked was there any hypopyon? In cases over 30 years of age he generally extracted the lens. Patients with diabetes do not stand needling well, but bear extractions much better. Trouble came after discission more often than after extraction, due perhaps to lens matter left behind.

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3. *Coppock cataract.*

By L. Werner.

Patient was a man, æt. 22 years. The cataract was bilateral. V. = $\frac{6}{12}$, R. and L. No history of eye trouble in other members of the family.

Mr. Werner thought this was the first case of its kind shown in Ireland.

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4. *Neoplasm of the cornea in elderly man thought to be epithelioma.*

By F. C. Crawley.

Meeting held at the Royal Victoria Eye and Ear Hospital, Dublin, March 23rd, 1922.

5. *Discussion on stricture of the nasal duct.*

Mr. T. O. Graham

opened the discussion and said that to overcome epiphora various operations have been devised, which have for their object the short-circuiting of the tears directly into the nose from the lacrymal sac. Toti advocated the transplantation of the outer wall of the sac bearing the entrance of the canaliculi into the outer wall of the nose. This he did by external operation.

West, however, conceived the idea of attacking the lacrymal sac from the nasal cavity, thus dispensing with the external skin incision. I have adopted his method and have modified it. The operation is most easily carried out under local anæsthesia. The area on the outer wall of the nose anterior to the middle turbinal is anæsthetised with the local application of 20 per cent. cocaine and adrenalin, or submucous injection of novocaine may be employed.

With a septum knife or small scalpel the mucous membrane about half-an-inch square is removed from the area. The underlying bone is then chiselled away, the window in the bone being enlarged by means of nibbling forceps. In this way the inner wall of the lacrymal sac is exposed, and can be invaginated into the nose by passing a probe through the lower canaliculus into the previously cocainised sac. While the probe thus invaginates the inner wall of the sac a vertical incision is made into its anterior surface with a fine-bladed sharp knife. The inner wall of the sac can then be removed with a conchotome. I find it unnecessary to use the special sharp-toothed tenaculum forceps for catching the sac. This completes the operation; the after-treatment consists in irrigating the sac through the canaliculus and preventing the formation of granulations around the artificial opening in the sac.

The difficulties encountered during the operation are:

(1) Thick bone, particularly a prominent sclerosed nasal process of the superior maxilla.

(2) An overlying anterior ethmoidal cell which may be opened up. One finds, then, that two layers of bone have to be removed in order to reach the sac, which lies at the bottom of a recess in the lateral wall of the nose.

(3) An enlarged anterior end of the middle turbinal which may require removal.

(4) Hæmorrhage during the operation. The operation may have to be completed under the sense of touch, a finger pressing into the lacrymal groove externally acting as a guide.

(5) A narrow nose. In order to obtain sufficient space for approaching the sac it may be necessary to do a preliminary submucous resection of the nasal septum.

I have endeavoured to ascertain by observation and inquiry by letter the end-results of the thirty intranasal dacryo-cystostomy operations I have performed during the past three years. Twenty-two have been complete successes. Four had lacrymal sac fistula prior to operations. All healed up after the intranasal drainage of the sac.

Four write to say they still have epiphora; two, however, admit improvement; one says there is now only watering of the eye and no purulent discharge.

Four cases have not replied to the letters of inquiry. Two of them were, however, free from trouble on leaving Dublin several weeks after the operation.

All these thirty cases have been referred to me for operation by ophthalmic surgeons who had carried out conservative treatment without permanent benefit.

Mr. L. J. Curtin

said his experience was not extensive, but the operation seems to present some difficulty from the operator's point of view.

(1) If the nose is narrow it is difficult to orientate properly owing to the lateral view obtained of the site of operation.

(2) Sufficient space is not always available for grasping and cutting instruments to be inserted at the same time.

These obstacles can be overcome to some extent by performing a high submucous resection of the septum or by following the transeptal route.

After-treatment is all-important, and it must be carried out from six to eight weeks following operation, with an occasional visit up to a later period. Granulations need removal when they make their appearance.

Mr. L. Werner

asked what were the advantages of the operations over excision of the sac. He thought two disadvantages were the difficulty of the operation and the slower healing. If the tears were drawn along by suction the operation might remove this. The liability to infection from the nose seemed to him considerable.

Mr. J. A. Craig

thought discussion was to some extent futile, as rhinologists and ophthalmic surgeons had their own opinions. The drawback to the operation is that frequently the eye still waters. The difficulty of the operation depends on (*a*) narrowness of nose; (*b*) difficulty of getting in two instruments to grasp the sac and cut it. He asked which would a rhinologist do in a case of caries on lupus?

Mr. H. C. Mooney

asked, Would a rhinologist refuse to operate in a necrotic case?

Mr. F. C. Crawley

inquired what were the indications for the operation. He would excise the sac previous to an operation for cataract. If the patient was a working man he would excise the sac and let him put up with the epiphora.

Mr. J. B. Story

had seen all the various methods of treatment and good results from all. The intra-nasal operation was only

indicated in cases of dacryocystitis, and only superior to excision of the sac in that you should not have epiphora as you have after excision. Two cases he had seen gave a perfect and permanent result after Maddox's treatment of injecting quicksilver. Excision was much the easier operation, and unless the intra-nasal operation produces more cures it would not come into use.

Mr. Graham,

in reply, said that cases to operate on by the intra-nasal method were: (1) Where ophthalmic surgeons had failed; (2) many cases where a fistula exists. Simple cases did better with ophthalmic treatment. The operation did not interfere with the physiological action of suction of the tears. A good result should be completely free from epiphora. He had had only one case of secondary infection from the nose.

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6. *Calcareous film of the cornea after sympathetic ophthalmia in* 1915.

By J. B. Story.

7. *Case of tumour of pituitary and region of basilar process of occipital bone.*

By L. Werner.

8. *Case of skin graft.*

By L. Werner.

Graft consisted of large piece of skin in its whole thickness. No antiseptic was applied to skin before removal. A dressing was boric acid freely dusted on: it is important to keep area dry.

9. *Lacrymal gland tumour treated by X rays and improving.*

By Miss Maxwell.

10. *Case of post-neuritic atrophy of each optic nerve in young man.*

By W. C. MacFetridge.

Ætiology unknown. Examination of blood, urine, fæces, expectoration, nasal sinuses was negative. There was a history of sand-fly fever.

OPHTHALMOLOGICAL SOCIETY OF EGYPT.

1921–22.

REPORTS.

1. *A case of cyst of the left orbit of a possible filarial nature (Filaria Bancrofti).*

By M. Sobhy Bey.

A male, æt. 30 years, presented himself at No. 1 Travelling Ophthalmic Hospital, Rod el Farag, on July 18th, 1921, with L. exophthalmos. He was of a fairly good constitution, and had never been out of Egypt. He noticed the bulging of his L. eye two years previously. A year later he received a blow with a fist which resulted in a black eye. After some time the traumatic swelling disappeared, leaving the eye more proptosed than before. No history or signs of syphilis, and no chyluria or hæmaturia.

Clinical description.—The case was first seen by Dr. Halim Abu Seif, the principal medical officer of Rod el Farag, who made the following notes: "T. III, peripheral pannus, pupils active. R.V. $\frac{6}{12}$, good fundus. L.V. $\frac{6}{36}$. T.n. The eye is markedly proptosed and pushed down and in. Upper lid when closed completely covers the globe. The fundus after dilatation of the pupil reveals slight hyperæmia of the disc and engorgement of the veins, but no hæmorrhages. A soft, tender, fluctuating mass could be felt at the outer side of the orbit separating the outer wall of the latter from the globe, which is thus pushed down. The skiascopic result, under homatropine and cocaine, was, for the R. eye, sph. + 2, cyl. + 0·5 ax. v., and that of the L. eye was sph. + 5, cyl. + 0·5 ax. 60."

On July 25th the case was admitted to Giza Ophthalmic Hospital. On the same day the blood was examined for Wassermann's reaction, with a negative result; nevertheless the patient was given 3 grm. of potassium iodide for three days.

On August 10th potassium iodide 2 grm. three times a day.

On August 13th potassium iodide 3 grm. three times a day.

On August 18th L. proptosis the same; no improvement; and potassium iodide was continued.

On August 21st L.V. $\frac{6}{36}$, and a note was made by Dr. MacCallan, who recommended taking the fields. These were found similar and of normal limit. The patient was then sent to Kasr el Ainy for skiagram of his skull. In the plate there was more opacity in the left orbit than the right. Kronlein's operation was advised and done at Kasr el Ainy by Mr. Dolbey in my presence. The external carotid was tied as a preliminary measure. The flap containing the integument and the malar bone was turned aside as usual, and after exposing the contents of the orbit the wall of the tumour was accidentally ruptured and a cheesy material poured out. This was at once collected aseptically in two sterile test-tubes for pathological examination. After evacuating all the contents of the cyst, Mr. Dolbey examined the seat of the cyst, and stated that it was communicating with the brain through the sphenoidal fissure, and thought it was possibly a case of psammoma of the temporo-sphenoidal lobe. At that moment I thought of inspissated suppurating hydatid or a meningocele. The operation was concluded, and was followed by marked improvement both in the vision and in the degree of proptosis. The cheesy matter was then submitted to a systematic laboratory research. Bits were fixed in Bouin's solution and prepared for cutting and staining. Sections have shown it to be an amorphous mass devoid of cellular elements except a few leucocytes. They indicate that degenerative processes are advanced. The Pathological

Department of Kasr el Ainy gave the same result. Then the cheesy material was inoculated on different media to find any germ, and again these proved to be sterile. As tuberculosis was suspected, glycerin carrots tubes were also inoculated, with a negative result. Before giving the case up, I made a smear from the deposit in an inoculated bouillon tube. This was stained, and a microfilaria was seen on the slide. This was shown to Dr. Hindle, Professor of Parasitology at the School of Medicine, who agreed that it was an embryo of *Filaria Bancrofti*. Then the patient's blood was examined at night time, and found to contain microfilariæ in large numbers. The patient was sent back to Kasr el Ainy and put under the care of Dr. Day, and had a full course of tartar emetic.

On January 12th, 1922, the patient was sent back to Giza Ophthalmic Hospital as cured. R.V. $\frac{6}{9}$, L.V. $\frac{6}{18}$ with cyl. + 1 ax. 115° down and out = $\frac{6}{12}$.

On January 23rd I examined the blood again, and found that the microfilariæ were less numerous than before.

Interpretation of the case.—The geographical incidence of the case makes the Loa Loa infection impossible; moreover the embryos look like those of *Filaria Bancrofti*.

Can we diagnose this cyst as being filarial in nature? If not, what could it have been? How would a filarial cyst form? This is better explained by the two following suppositions: (1) An adult filaria, where it lives or where it dies—as a rule in the lymphatics—produces a dilatation of the latter with damming up of the lymph and cyst-like formation. This is frequent in the thoracic duct, but it may happen anywhere in the body where there are lymphatics. (2) The trauma incurred by the patient might have brought about the death of a filaria worm already present in the orbit, with the result of inflammation of the tissues around, sterile suppuration and then inspissation of the pus. One might contend that if the cyst was really the seat of a filaria worm one ought to get more microfilariæ in the cyst than in the blood.

But if there has been a living worm in the cyst, the embryos should not remain in the lymph-stream (*i. e.* the cyst as well), as they are wanted in the general circulation ready to be sucked by the culices. (This explains the fact that in many cases of chyluria microfilariæ are absent.) Another possibility is that the microfilaria found in the cheesy matter was there only through contamination from the patient's blood during the operation.

Here again if the adult worm had died there would be no formation of embryos in the cyst. It is to be borne in mind that not infrequently in filariasis there is more than one worm in the same host, and so if one dies it is not strange to see the host sill harbouring microfilariæ. Moreover, the filariasis continues to show itself up to six months after the death of the worm.

The structureless character of the sections rules out meningocele. The walls of the cyst do not look like those of hydatid. Had the cyst orginated in the brain it would have given rise to symptoms of intracranial pressure which were completely wanting in the case.

Taking all things into consideration one is justified, I believe, in attributing the cyst in question to filariasis.

In conclusion, I thank all my colleagues who have assisted in this unique case.

2. *Appearance of the fundus in ankylostoma worm infection.*

By M. Riad.

I have requested my colleague Dr. Erfan, the Registrar at Kasr el Ainy Hospital, to send me a few cases of severe anæmia due to ankylostoma worm infection for fundus examination. He was kind enough to send me twenty cases in all.

In seven cases the percentage of hæmoglobin varied between 30–50 per cent., and the rest between 60–70 per

cent. In the first lot the choroidal reflex was of a light yellow colour instead of being rich red; veins large, tortuous, flabby, and had the appearance of flattened ribbons; arteries were thin and in a few cases the retinal arterioles were corkscrew-shaped.

In one case I. H— (Hospital No. 1 893), the superior and inferior retinal arteries were pulsating. This is explained by the fall of arterial pressure in which the central retinal artery takes part.

In one case, A. G— (Hospital No. 2753; hæmoglobin 40 per cent., a few white dots were observed along the right superior temporal artery which may have been fibrinous masses of exudate, and two fresh flame-shaped hæmorrhages were present in the inferior temporal quadrant two disc diameters away from the disc. These hæmorrhages may be due to alteration of the composition of the blood affecting the walls, and to the action of toxins introduced into the body by the parasite.

The most interesting case of all is the case of a negro, A. K. A. M—, æt. 50 years (Hospital No. 877), in which the amount of hæmoglobin was 30 per cent. My notes about him run as follows: both eyes are emmetropic, R. vision $\frac{6}{24}$, L. $\frac{6}{18}$; T. normal. Right fundus tigroid appearance, choroidal reflex pale, veins dilated, tortuous and pulsating, arteries thin, disc very pale with sharply defined edge. Left fundus same appearance as right, but disc is not so pale. Fields taken for white in good daylight 10 mm. disc show contraction of both fields, especially on the nasal side of R. field. Wassermann not performed, but no family or personal history justifies the suspicion of syphilis.

The pallor of both discs might be diagnosed as anæmia of the disc capillaries, but this is rejected by :—

(1) The defective central vision and the contraction of the fields.

(2) The asymmetry in the colour of both discs.

Inayuti of Japan reported a case of retro-bulbar neuritis due to ankylostomiasis, but as far as I can gather

no case has been recorded of primary optic atrophy in this particular disease. In the other cases of higher percentage of hæmoglobin the fundi appeared normal except for the dilatation in the veins and slight paleness in the colour of the choroidal reflex.

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3. *Report on result of treatment of the case of arterio-venous aneurysm of orbit reported to this society last year.*

By M. Tewfik.

Last year I reported to this Society a case of pulsating exophthalmos of the right eye due to traumatic arterio-venous aneurysm from gunshot wound in a young boy, M. Y—, æt. about 15 years.* The boy was operated upon in March, 1921, by Mr. Dolbey, surgeon at Kasr el Ainy Hospital, by ligaturing the common and external carotid, the latter being done to avoid collateral circulation. The swelling diminished perceptibly on the table and a deep suture was passed through the main bulk of the swelling and tied.

The boy was again recently examined by Dr. Girgis, who reported that no tumour could be detected now; the operation-scar is visible over the neck.

I must express my thanks to the operating surgeon, Mr. Dolbey, and to Dr. MacCallan and Dr. Girgis in helping me to get at the patient now.

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4. *A case of palpebral sporotrichosis (Lymphangetic form).*

By M. Sobhy Bey.

The patient, No 30617, Stationary Ophthalmic Hospital, Giza, a male, æt. 30 years, laundryman, was sent to me on March 26th, 1921, by Dr. Barrada, ex-dermatologist at Kasr el Ainy Hospital.

Past history.—A few years ago the patient had an ulcer

* *Trans. Ophth. Soc. U.K.*, 1921, vol. xli, p. 528.

on the genitals followed by an eruption which was accompanied by itching. Six months ago the patient had redness and swelling of the left eye with discharge. This condition remained for a week and was treated by some kind of lotion and drops. The eye has been quite healthy since then.

Present history.—A married man of moderate life, of a fair constitution and of medium height and body. Six weeks ago the patient noticed a swelling at the inner canthus, which shortly was followed by a swelling of the upper and lower lids. The patient then sought the advice of Dr. Galal, of Qalawoon Ophthalmic Hospital, who ordered him an ointment and hot bathings. After a week's treatment the swelling at the internal commissure ulcerated and Dr. Galal, suspecting its specific nature, sent his patient to Dr. Barrada for an opinion.

Description.—A shallow ulcer is seen on the skin just below the lower canaliculus of the left eye, 3 mm. by 8 mm. in area, with a brawny swelling around, giving a sensation of hardness; the ulcer itself feels soft. This brawny swelling spreads up and down in a concentric manner round the palpebral aperture, the outer commissure being free from œdema. In the skin of the lower lid there is a nodule as big as a pea, the upper border of which touches the lower border of the tarsus. There is another swelling in the upper lid similar to the lower one, a little smaller; the skin over these nodules is œdematous and does not move over them. There is a cord-like resistance from the upper nodule going to the ulcer. There is a similar one in the lower lid that can hardly be felt. The preauricular gland is a little enlarged. Temperature was 36·8° C. and showed no rise later. A well-marked sore on genitals is present. Enlarged glands can be felt all over the body except on the occipital region. The throat was red and injected, but there was no ulceration. Patient was addicted to smoking. He was seen again on March 27th, 1921, in the Ophthalmic Laboratory, Giza. Smears and cultures were taken from the ulcer before

cleaning it, and from the discharge covering its base, on serum agar, blood agar and simple agar. The ulcer was then cleaned with sponges, freeing its base from the discharge, and other slides were taken and other tubes were inoculated. To irritate the ulcer in order to obtain a serous discharge for the examination of Schaudinn's spirochæte, according to the method of Bury, by Indian ink, it was rubbed with a sponge impregnated with alcohol. A Pravas syringe was then prepared and the needle was introduced into the lower nodule after painting it with iodine tincture. A very small quantity of pus was obtained and this was smeared on the surface of a simple agar tube. This last tube was put in the incubator. The blood was examined at the Public Health Laboratory for Wassermann's reaction and was found positive.

Report of the laboratory examination.—The first slide, taken directly from the ulcer, was stained with methylene blue and a long branching mycelium with two conidiæ attached to it was found. A more prolonged research revealed nothing of the sort in all the other slides except two bodies looking like spores (or conidiæ). The three slides prepared according to Bury's method were exhaustively examined and the result was negative for the *Spirocheta pallida*. I stained more slides with the slow and quick methods of Giemsa and no spirillæ could be found. The tubes inoculated were examined from time to time. Some of these remained quite sterile, while others only showed a few growths of cocci or some yeast colonies and one colony of mycelium. (This latter was of a clear chocolate colour, rounded, 5 mm. in diameter and 2 mm. high, of an irregular edge and a surface showing protuberances and convolutions. It was not easy to make a smear from it as it was difficult to get through it with the needle and it seemed very adherent to the media. A penetration of this colony in the media could not be made out as the latter was not clear, being a blood agar. A smear did show a branching mycelium of a voluminous size. The threads of the mycelium were ribbon-like, *i.e.*

flat. Their branching was at an obtuse angle, and in general they did not look at all like the threads of sporotrix.) The agar tube, inoculated with the pus aspirated from the nodule and kept in the incubator, was examined on the fifth day. Besides one colony of a *Staphylococcus albus* there were strewn on the surface of the agar other small ones, with a smooth rounded surface which had a defined edge. They looked clear at first, becoming opalescent when old. A smear was made from these little colonies and was stained with methylene blue. I could see short threads or short stems at their bifurcation into two or more. The threads showed septa enclosing short spaces. The spaces took lightly the stains as if they were spores. The mycelium looked either straight or beaded from the swelling produced at the clear spaces. The conidiæ were single, *i.e.* one at the end of each thread, or free. (The free conidiæ were round, circular in shape and not oblong, surrounded with their capsule, which seemed to take the blue stain of methylene blue while the inside was pale. Another interpretation of this character, the peripheral part of the protoplasm took the stain, the inside and the capsule remaining clear. This description is different from that given to the two varieties of sporotrichosis, Shenki and Beurmannii, where the conidiæ are multiple and oblong, and these take the stains readily.) The tube was then taken out of the incubator and left at the room temperature. The colonies increased in number, coalesced, but never changed their colour, and remained always of a white opaque colour. This made me classify this strain in the variety of sporotrix described by Dor or an allied strain. Proper glucose and glycerine media were then prepared, and subcultures were tried on these as well as on blood and serum media on many occasions, and all efforts remained futile. Anaërobic cultures have failed.

Experiments on animals.—A thin emulsion of the fungus was prepared and injected under the skin of the ear of a young rabbit. Some of the pus aspirated from

the nodule was injected under the skin of a guinea-pig to exclude the presence of tuberculosis. The rabbit was kept for months under observation and showed no lesions; the guinea-pig disappeared from its cage. Subcultures from the original tubes were tried again on glycerin glucose bouillon which was left at the room temperature. A very thin film appeared on the surface of the bouillon with some turbidity of the latter. This showed again the presence of the fungus. The blue litmus bouillon media turned a faint red, then bleached with the absence of gas formation. These cultures will be used again for the animal inoculations.

Diagnosis.—Clinically we were confronted with an ulcer accompanied by lymphangitis. It is true any ulcer (septic) might be followed by a lymphangitis. This is more common with syphilis, tubercle, glanders and sporotrichosis. The ulcer was not typically hard. Taking the observation of Dr. Galal as granted, I doubted the possibility of its being venereal as in these cases the lymphangitis comes on after the ulceration and not before. In other words, the lymphangitis seen in the venereal disease is not specific—spirochetæ do not form lymphangitis, it is due to mixed and secondary infection, as in phagedenic ulcers, etc. I at once thought of the possibility of the lymphangitic form of the sporotrichosis; tubercle and glanders did not seem probable. However, the microscopic examination and the course of the case under treatment decided the diagnosis.

Treatment.—These cases do badly if they are treated surgically, and one is warned against using the scalpel to open the nodules or abscesses formed by this kind of fungus. The iodine treatment is specific either in the free form or as potassium iodide. On April 3rd potassium iodide was ordered in the dose of 2 grm. daily, which was increased every three days. Improvement appeared from the first week. On April 23rd 10 grm. of potassium iodide were being given daily. The ulcer healed and the swelling of the upper and the lower lids diminished in

size. On April 25th potassium iodide was stopped. On May 4th potassium iodide was re-ordered, 2 grm. daily for a fortnight, and then stopped altogether.

The object of this long course of potassium iodide was to prevent any recurrence. On June 19th the patient showed no signs of his disease except some browny pigmentation of his lids, there were no signs of recurrence. Wassermann remained positive after all this potassium iodide treatment, which suggests that this drug has no curative effect on syphilis.

The patient was advised to undergo a complete mercurial and arsenical treatment for his syphilis.

As far as I am aware this is the second* case of sporotrichosis ever published in the medical literature, or proved by bacterial examination in this country. In general surgery and medicine this disease may not be very rare, but they are rare in ophthalmic practice.

After writing the above lines, animal inoculation was undertaken on a rabbit from a culture on glucose bouillon. Injections were made under the conjunctiva as well as in the region of the lacrymal sac, with negative results. After many subcultures with simple bouillon and glucose bouillon the organism took a somewhat different picture than that already seen in the first cultures: the mycelium is longer, straighter and more slender, and stains uniformly without showing the beaded appearance already described. As to the spores of the conidiæ they show two forms—one which takes the stain more or less uniformly with some pallor in the centre, the other takes the stain only at the periphery while the centre remains colourless. Here and there one of the spores is seen bearing a stalk.

Before closing I want to emphasise the fact that streptothrix and sporotrichosis are two quite different conditions of the conjunctiva.

* The first case was that of sporotrichosis of the tongue, published by Duverger, of the French Hospital of Cairo, and cultures obtained by Bain, of the Institut Pasteur, photographs made by Dr. Hamid Ouasef.

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Morax and Carlotti.—"La sporotrichose palbebrale," *Annales d'Oculistique*, 1908, C, xxxix, 418. (The English *resumé* found on p. 973 of *The Ophthalmoscope*, December, 1908.)

English readers are also recommended to read what was written on Sporotrichosis: *Ophthalmoscope*, 1909, p. 555; 1911, p. 195; 1914, pp. 222, 299, 679; 1915, p. 392 from *The Annals of Ophthalmology*, October, 1914.

La Sporotrichose, form lymphangitique, thèse de Lyon, par Hussein Zohdi, 1909.

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5. *Ocular complications occurring in malaria.*

By W. H. Kiep.

I have taken the liberty of introducing the subject of the ocular complications of malaria, not so much with a view to imparting information as with the object of obtaining it. The subject is one with regard to which the greatest diversity of opinion imaginable exists. Thus Poncet (1), writing in 1878 of his experience in the north of Africa, states that eye complications occur in 10 per cent. of cases of malaria, whilst more recently (in 1919) Kirkpatrick (2), at that time in Madras, stated "I have not been able to assign malaria as a cause of any fundus disease, although I think it is sometimes the cause of . . . dendritic ulcer of the cornea, or some superficially placed interstitial keratitis." Elliot, in his *Manual of Tropical Ophthalmology* (3)—the first text-book which attempts to deal in a systematic fashion with ocular complications of tropical diseases—expresses himself as somewhat sceptical of Poncet's findings, and later on states, "There is plainly a conflict of opinion as to the true aetiology of retinal haemorrhages in malaria," that is, as to whether these haemorrhages are actually caused by the malarial parasite or are simply due to the severe anæmia that occurs in many cases of the disease. Perhaps I may

be excused if I state my own experience in the first place. During the years 1916, 1917 and 1918 I happened to have a large number of troops suffering from ocular disease and also infected with malaria under my charge. These cases had all been infected in Macedonia, and included cases of infection with *Plasmodium vivax*, and others infected with *Plasmodium falciparum*. In brief, the ocular conditions occurring in malarial patients, which I attribute to the malarial infection, are the following :—

(1) *Dendritic, or herpetic keratitis.*

(2) *A deep-seated central parenchymatous keratitis*, in my experience always unilateral—called by Fuchs (4) "*Keratitis Profunda*," and by Maxwell (5) "Discoid Keratitis."

(3) *Retinal and pre-retinal hæmorrhages.* These may in some cases give rise to new formation of connective tissue, either in the retina itself, as pointed out by J. Herbert Fisher (6), or in the vitreous, causing *retinitis proliferans.*

(4) *Optic neuritis*, generally accompanied by retinal hæmorrhages, sometimes quite definitely not so accompanied.

(5) *Lagophthalmos, i.e.* paresis or paralysis of the seventh cranial nerve.

In passing I may state that I have not been able to make up my mind that malaria was the cause of any case of uveitis I ever saw, although it has been shown by Dudgeon (7) that the malarial parasite was present in large numbers in the vessels of the iris of a case of malignant malaria that he examined post mortem.

With regard to (1) *dendritic or herpetic keratitis* :—

It is, I think, fairly generally agreed now that this condition may be brought about by malaria, just as, apparently, it may be produced by influenza, cold in the head, and other pyrexial conditions. It is, of course, quite a separate condition from herpes zoster ophthalmicus, just as herpes labialis (or facialis) is a condition different from herpes zoster generally (8). It is interesting to note

that 22 per cent. of my cases gave a definite history of herpes facialis (not herpes zoster ophthalmicus). In all I saw 63 cases of keratitis herpetiformis, and of these, 46, *i.e.* 73 per cent., gave a clear history of no previous eye trouble. The ocular condition might arise on the same day as the initial malarial attack, or long after it. In my experience the condition is invariably unilateral. In most cases in which data were available the infection was with *Plasmodium vivax*, but in one case it was quite definitely due to the parasite of malignant subtertian malaria.

(2) The other form of keratitis which I have alluded to is an interstitial, *i.e. parenchymatous* form, the corneal epithelium not being affected in the ordinary course of events. Altogether I saw four cases of this condition. In each one the Wassermann test was negative, and careful examination failed to reveal the presence of any of the stigmata of congenital syphilis. Moreover, in every case the keratitis was unilateral. In each case the parasite of benign tertian fever was found in the blood. This type of keratitis progresses extremely slowly in my experience, the duration of the inflammatory period averaging three months, and a permanent corneal opacity is left, which, being centrally situated, gives rise to a good deal of interference with vision. In one case that I had the opportunity of observing over a period of two years, the central scar was still present at the end of that time, vision being reduced to less than $\frac{6}{60}$, although all signs of active inflammation had long since disappeared from the eye.

(3) *The occurrence of hæmorrhages in or from the retina* in cases of malaria is fairly generally conceded, but their exact causation has for long been a matter of doubt. Thus Edgar Smith (9), working at Salonica during the war, rather tended to the opinion that they occurred only in cases in which the red cell count was below 3 million cells per c.mm., and in which the cause might quite readily have been anæmia only. More recently, however, Dudgeon (7) has shown that the malarial parasite may be

present in the vessels of the optic nerve and retina, and it is probable, as Herbert Fisher suggests (6), that the true cause of the retinal hæmorrhages is to be found in the blocking of the respective vessels with the malarial parasite. Fisher himself has published a case of connective-tissue formation in the macular region, which, he believes, resulted from the organisation of a film of blood extravasated from an obstructed retinal vein. My own experience tallies exactly with this.

In all I saw some ten cases, the nature of which may very well be explained by the occurrence of hæmorrhage from obstructed retinal veins—six cases of hæmorrhage only, including one of subhyaloid hæmorrhage and two others in which hæmorrhage into the vitreous had occurred and had been followed by retinitis proliferans. The other four of these ten cases showed optic neuritis in addition to retinal hæmorrhages. It is interesting to note that while some of these cases appeared to be due to the parasite of malignant subtertian malaria, others showed the presence of *Plasmodium vivax* only in the blood.

(4) In addition to these I saw seven cases of *optic neuritis* (in all cases except one, bilateral) without hæmorrhages, and two cases of *post-neuritic atrophy* both of which gave a negative Wassermann reaction and had retinal arteries of normal calibre, so that it would appear that these cases of atrophy were due neither to syphilis nor quinine.

(5) Lastly, I should like to bring before your notice four cases of *lagophthalmos*, which I attribute to malarial infection. One of these had had cerebral malaria, and in addition to lagophthalmos there was nystagmus. These manifestations only commenced when convalescence from the attack of cerebral malaria was well established. Another case developed facial paralysis some weeks after an attack of benign tertian malaria, and was interesting in that a subsequent attack of ague considerably aggravated the facial paralysis.

I fear that my descriptions of the various conditions has

been somewhat superficial, but in the limited time at my disposal it has not been possible to go into the subject more thoroughly.

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(2) Kirkpatrick. — Quoted by Elliot in *Tropical Ophthalmology*, p. 448.

(3) Elliot.—*Tropical Ophthalmology*, Oxford Medical Publications, 1920.

(4) Fuchs.—*Text Book of Ophthalmology*, English Translation by Duane, p. 282.

(5) Maxwell. — *British Journal of Ophthalmology*, August, 1918.

(6) Fisher, J. Herbert.—*Trans. Ophth. Soc. U.K.*, 1921, vol. xli, p. 235.

(7) Dugdeon.—*Trans. Ophth. Soc. U.K.*, 1921, vol. xli, p. 237.

(8) Norman Walker.—*An Introduction to Dermatology.*

(9) Smith, H. E. — Quoted by Elliot in *Tropical Ophthalmology*, p. 455.

Dr. S. A. H. Soliman.—It is not enough to exclude syphilis, but tubercle also must be excluded by Von Pirquet's test before ascribing these cases of parenchymatous keratitis to malaria.

Dr. M. A. Bakly.—I may here report the case of a soldier, sent to me at Asswan from Halfa, who had an attack of malaria, and was given quinine injections. Though cured of his malaria, the poor man totally lost his vision. Fundus examination showed an atrophied disc with slightly contracted vessels. Was the atrophy due to the effect of quinine or was it caused by the malaria? I could not decide.

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